J. Nat. & Phy. Sci. Vol. 21 2007 No. 1-2

132092

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132092

ISSN 097-3799

प्राकृतिक एवं भौतिकीय विज्ञान शोध पत्रिका

JOURNAL OF NATURAL & PHYSICAL SCIENCES

खण्ड 21 अंक 1-2, 2007
Vol. 21 No. 1-2, 2007

गुरुकुल कांगड़ी विश्वविद्यालय, हरिद्वार
Gurukula Kangri Vishwavidyalaya, Haridwar

प्राकृतिक एवं भौतकीय विज्ञान शोध पत्रिका

Journal of Natural & Physical Scienes

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हस्ताक्षर
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कुलसचिव

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132092

Printed at : Kiran Offset Printing Press, Gurukul Kangri, Haridwar, Phone : 01334-245975

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 1-9

GLOBAL STABILITY OF THREE-SPECIES PREDATOR PREY SYSTEM

I.H. Elmabruk

(Received 16-05-2007)

ABSTRACT

A model of three level food web, as given in E*l*- Owaidy and Ammar [6] is extended with density dependent death rates and then global stability of equilibrium state is discussed by constructing Lyapunov function of the system.

Keywords. : Predator prey-Global stability Equilibrium Lyapunov function-Response function.

INTRODUCTION

The question of global stability in predator prey system is a very interesting problem both from mathematical and biological point of view. It is quite interesting to find conditions which may guarantee the global stability of the unique positive equilibrium.

The global stability analysis for the classical Lotka-Volterra system has been given by Goh [2] by constructing a Lyapunov function. Hsu [4] has given a Lyapunov function that works for a class of models that have a non linear functional response. But Hsu [4] employed the Bendixson-Dulac criterion to prove global stability for an intermediate type predator-prey model as discussed by Freedman [1] and Liou and Cheng [5]. Harrison [3] gave a Lyapunov function that extends functions used by Volterra, Goh and Hsu to a wide class of predator-prey models.

In this work, we have constructed a Lyapunov function on the line of Harrison [3] gave a Lyapunov function that extends functions on the line of Harrison [3] for an intermediate type 3-species prdedator-prey model and have given conditions which guarantee the global asymptotic stability of unique positive equilibrium.

THE MODEL

We consider the intermediate class of 3-species predator prey model

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*Department of Mathematics, Faculty of Science, University of Garyounis, Benghazi, Libya.

$$\begin{aligned}
\dot{x} &= xf(x) - yp(x) - zq(x) \\
\dot{y} &= y[g(y) + c_1 p(x)] - zr(y) \\
\dot{z} &= z[-h(z) + d_1 q(x) + d_2 r(y)] \\
x(0) &= x_0 \geq 0,\ \ y(0) = y_0 \geq 0, z_0 \geq 0,
\end{aligned} \tag{2.1}$$

where $\left(\bullet = \dfrac{d}{dt} \right)$, the functions $f, p, q, r, g, h,$ are suffifiently smooth so that the solution to the initial value problem (2.1) exists, is unique and continuable for all positive values of t and c_1, d_1, d_2 are positive constants.

We think of $x(t)$ as a prey population $y(t)$ as a prey as well as a predator population and $z(t)$ as a predator population at a given time $t \geq 0$.

The following assumptions are consistent with models of predator prey systems for x, y, $z \geq 0$ and with an assumption that the environment has a natural carrying capacity.

(H1) : $f(x)$; specific growth rate of prey x

$f(0) > 0$, $f'(x) > 0$ for all $x > 0$, there exists $k_1 > 0$ (the environmental carrying capacity) such that $f(x) > 0$ on $0 < x < k_1$, $f(k_1) = 0$, $f(x) < 0$ on $x > k_1$

(H2): $p'(x)$: the first predator function $p(0) = 0$ and $p^1(x) > 0$, for all $x > 0$.

(H3) : $q(x)$; the second predator response function $q(0) =$ and $q'(x) > 0$, for all $x > 0$.

(H4) : $r(y)$: the second predator response function when the first predator is considered as a prey $r(0)$ and $r'(y) > 0$, for all $x > 0$.

(H5) : $g(y)$ specific growth rate of prey y

$g(0) >, g'(y) \leq 0$ for all $y > 0$, there exists $k_2 > 0$ (a carrying capacity) such that $g(y) > 0$ on $\leq y < k_2$, $g(k_2) = 0$, $g(y) < 0$ on $y > k_2$.

(H6) : $H(Z)$ is the density dependent predator death rate

$h(0) > 0, h'(z) \geq 0$, for all $z > 0$.

Remark 2.1:

Our model (2.1) is an extension of the model (1.1) of El-Owaidy and Ammar [6], in a sense we have used density dependent death rates.

In [6], local stability of equilibria was discussed, whereas we have discussed, the global asymptotic stability of the equilibria by constructing the Lyapunov function, Clearly, the system (2.1) has equilibria at

$E_0(0,0,0),\ E_1(k_1,0,0),\ E_2(0,k_2,0).$

$E_3(\bar{x},\bar{y},0)$ in the first quadrant of the *x-y* plane,

$E_4(\hat{x},0,\hat{z})$ in the first quadrant of the *x-z* plane, and

$E_5\,(0,\dot{y},\dot{z})$ in the first quadrant of the *y-z* plane,

The questions of whether or not there exists an equilibrium in the interior of the first octant, is also of interest. We assume there exists such an equilibrium which is positive and we label it *E*(*x,y,z*), moreover, it is a positive solution of the alghebraic system;

$$\begin{aligned}
&xf(x) - yp(x) - zq(x) = 0\\
&yg(y) + c_1 yp(x) - zr(y) = 0\\
&-h(z) + d_1 q(x) + d_2 r(y) = 0
\end{aligned}$$

THE LYAPUNOV FUNCTION

In this section, we construct the Lyapunov function in the following three cases.

3.1 Two preys x (t), y(t) and one predator z (t) :

The system (2.1) takes the form:

$$\begin{aligned}
&\dot{x} = xf(x(-zq(x)\\
&\dot{y} = g(y) - zr(y)\\
&\dot{z} = z[-h(z) + d_1 q(x) + d_2 r(y)]
\end{aligned} \tag{3.1.1}$$

Letting $E\,(x_1, y_1, z_1)$ be the unique positive equilibrium of the system (3.1.1) which is given by

$$\begin{aligned}
&x_1 f(x_1) - z_1 q(x_1) = 0\\
&y_1 g(y_1) - z_2 r(y_1) = 0\\
&-h(z_1) + d_1 q(x_1) + d_1 r(y_1) = 0
\end{aligned} \tag{3.1.2}$$

where it is assumed that

$$\frac{x_1 f(x_1)}{q(x_1)} = \frac{y_1 g(y_1)}{r(y_1)} \tag{3.1.3}$$

We define the Lyapunov function $v(x,y,z)$ for (3.1.1) as :

$$v(x,y,z) = d_1 \int_{x_1}^{x} \left[1 - \frac{q(x_1)}{q(w)}\right] dw + d_2 \int_{y1}^{y} \left[1 - \frac{r(y_1)}{r(w)}\right] dw + \int_{z_1}^{z} \left(\frac{w - z_1}{w}\right) dw \tag{3.1.4}$$

Considering the region

$$A_1 = \{(x,y,z) \colon 0 < x_1 < x < k_1,\ 0 < y < \gamma < z_1 < z < \beta_1\} \tag{3.1.5}$$

where γ_1, β_1 are positive constants, $i = 1, 2, 3$ and $\gamma_1 = k_2$

From (3.1.4), (H3) and (H4) it follows that $V(x_1, y_1, z_1) = 0$, and is positive at all other points in the region A_1 ($i = 1$, in (3.1.5))

The time derivative of $V(x,y,z)$ with respect to (3.1.1) is given by

$$\begin{aligned}\dot{V}(x,y,z) = {} & d_1[q(x) - q(x_1)]\left[\frac{xf(x)}{q(x)} - z\right] + d_2[r(y) - r(y_1)]\left[\frac{yg(y)}{r(y)} - z\right] \\ & + (z - z_1)[-h(z) + d_1 q(x) + d_2 r(y)]\end{aligned}$$

Using (3.1.2), and (3.1.3), we have

$$\begin{aligned}\dot{V}(x,y,z) = {} & d_1[q(x) - q(x_1)]\left[\frac{xf(x)}{q(x)} - \frac{x_1 f(x_1)}{q(x_1)}\right] + d_2[r(y) - r(y_1)]\left[\frac{gy(y)}{r(y)} - \frac{y_1 g(y_1)}{r(y)}\right] \\ & + (z - z_1)[h(z_1) - h(z)]\end{aligned}$$

Assuming $\dfrac{x_1 f(x)}{q(x)}$ and $\dfrac{yg(y)}{r(y)}$ to be both non - increasing with one of them is strictly decreasing, then the equilibrium $E_6(x_1, y_1, z_1)$ is globally asymptotically stable. Thus, we have proved the following theorem.

Theorem 3.1.1:

Assuming that (H1), (H3), (H4), (H5), (H6), hold for the system (3.1.1), and in the region A_1 given by (3.1.5) the functions $\frac{xf(x)}{q(x)}$ and $\frac{yg(y)}{r(y)}$ are both non- increasing with one of them strictly decreasing. Them the equilibrium $E_6(x_1, y_1, z_1)$ is globally asymptotically stable.

3.2 One Prey x (t) And Two Predatros y (t) And z (t)

The system (2.1) takes the form:

$$\begin{aligned} \dot{x} &= xf(x) - yp(x) - zq(x) \\ \dot{y} &= y[-g(y) + c_1 p(x)] \\ \dot{z} &= z[-h(z) + d_1 q(x)] \end{aligned} \qquad (3.2.1)$$

where, we have the following assumptions :

(H1), (H2), (H3) and (H6) as stated under the heading 'Model'

(H7): $g(y)$: the density dependent death rate of the predator y satisfying

$g(0) > 0,\ g^1(y) \geq 0$ for all $y > 0$

(H8) : $q(x) = ap\ \alpha\rho(x)$, where a is α positive constant.

Letting $E_6(x_2, y_2, z_2)$ be the unique positive equilibrium of the system which satisfies the following algebraic system:

$$\begin{aligned} & x_2 f(x_2) - y_2 p(x_2) - z_2 q(x_2) = 0 \\ & -g(y_2) + c_1 p(x_2) = 0 \\ & -h(z_2) + d_1 q(x_2) = 0 \end{aligned} \qquad (3.2.2)$$

We define the Lyapunov function for (3.2.1) as:

$$V(x,y,z) = \int_{x2}^{x} \left[c_1 \frac{p(x_2)}{p(w)} \right] + d_1 \left[1 - \frac{q(x_2)}{q(w)} \right] dw$$

$$+ \int_{y_2}^{y} \left[\frac{w - y_2}{w} \right] dw + \int_{z_2}^{z} \left(\frac{w - z_2}{w} \right) dw \qquad (3.2.3)$$

From (3.2.3) and due to conditions (H2) and (H3), it is clear that $V(x_2, y_2, z_2) = 0$ and it is positive at all other points in the region A_2 ($i=2$ in (3.1.5))

The time derivative of $V(x,y,z)$ with respect to (3.2.1) is given by

$$\dot{V}(x,y,z) = c_1[p(x) - p(x_2)]\left[\frac{xf(x)}{p(x)} - \frac{zq(x)}{p(x)} - y\right]$$

$$+d_1[q(x) - q(x_2)]\left[\frac{xf(x)}{q(x)} - \frac{yp(x)}{q(x)} - z\right]$$

$$+(y - y_2)[-g(y) + c_1 p(x)] + (z - z_2)[-h(z) + d_1 q(x)]$$

Using (3.2.2) and (H8), we have

$$\dot{V} = \left[c_1[p(x) - p(x_2)] + \frac{d_1}{\alpha}[q(x) - q(x_2)]\right]\left[\frac{xf(x)}{p(x)} - \frac{x_2 f(x_2)}{p(x_2)}\right]$$

$$+(y - y_2)\left[g(y_2) - g(y) - \frac{1}{\alpha} d_1[q(x) - q(x_2)]\right]$$

$$+(z - z_2)\left[h(z_2) - h(z) - \alpha\, c_1[p(x) - p(x_2)]\right]$$

Due to the hyptheses (H1), (H2), (H3), (H6) (H7), and if we assume that the function $\frac{xf(x)}{p(x)}$ is strictly decreasing in A_2 then $V(x,y,z) < 0$, that is $E_6\,(x_2, y_2, z_2)$ is globally asmptotically stable.

Thus, we have proved the following theorem.

Theorem 3.2.1 :

Assuming that (H1), (H2), (H3), (H6), (H7), hold for the system (3.2.1) and the function $\frac{xf(x)}{p(x)}$ is strictly decreasing. Then the equilibrium $E_6\,(x_2, y_2, z_2)$ is globally asymptotically stable.

3.3 A Three level food web:

A Model of three Level food web, where there is a prey x at the lowest, level which is the sole food source of predator y at the intermediate level, which in turn is a food source of predator

z at the highest level, the predator z also feeds on the prey x at the lowest level.

The system (2.1) takes the form

$$\begin{aligned}\dot{x} &= xf(x) - yp(x) - zq(x)\\ \dot{y} &= y[g(y) + c_1 p(x)] - zr(y)\\ \dot{z} &= z[-h(z) + d_1 q(z) + d_2 r(y)]\end{aligned} \tag{3.3.1}$$

Letting $E_6\,(x_3, y_3, z_3)$ be the unique positive equilibrium of the system (3.3.1) which is given by:

$$\begin{aligned}&x_3 f(x_3) - y_3(x_3) - z_3 q(x_3) = 0\\ &y_3 g(y_3) + c_1 y_3 p(x_3) - z_3 r(y_3) = 0\\ &-h(z_3) + d_1 q(x_3) + d_2 r(y_3) = 0\end{aligned} \tag{3.3.2}$$

We define a Lyapunov function for (3.3.1)

$$V(x, y, z) = d_1 \int_{x3}^{x} \left[1 - \frac{q(x_3)}{q(w)}\right] dw + d_2 \left[\int_{y^3}^{y} 1 - \frac{r(y_3)}{r(w)}\right] dw + \int_{z_3}^{z} \frac{(w - z_3)}{w} dw \tag{3.3.3}$$

From (3.3.3) and due to conditions (H3), and (H4), it is clear that $V(x_3, y_3, z_3) = 0$ and it is positive at all other points in the region A_3 ($i = 3$ in (3.15))

The derivative of $V(x, y, z)$ with respect to system (3.3.1) is given by

$$\dot{V}(x, y, z) = d_1[q(x) - q(x_3)]\left[\frac{xf(x)}{q(x)} - \frac{yp(x)}{q(x)} - z\right]$$

$$+ d_2[r(y) - r(y_3)]\left[\frac{yg(y)}{r(u)} + c_1 \frac{yp(x)}{r(y)} - z\right] + (z - z_3)[-h(z) + d_1 q(x) + d_2 r(y)]$$

Using (3.3.2) we have

$$\dot{V}(x, y, z) = d_1[q(x) - q(x_3)]\left[\frac{xf(x)}{q(x)} - \frac{x_3 f(x_3)}{q(x_3)} - \frac{yp(x)}{q(x)} + \frac{y_3 p(x_3)}{q(x_3)}\right]$$

$$+d_2[r(y)-r(y_3)]\left[\frac{yg(y)}{r(y)}-\frac{y_3g(y_3)}{r(y_3)}+c_l\,\frac{yp(x)}{r(y)}-c_l\,\frac{y_3p(x_3)}{r(y_3)}z\right]+(z-z_3)[-h(z)]$$

Due to the hypotheses (H3), (H4), (H5), (H6) and if we assume that the functions $\frac{xf(x)}{q(x)}, \frac{yg(y)}{r(y)}, \frac{yp(x)}{r(y)}$ are non - increasing and $\frac{yp(x)}{q(x)}$ in non - decreasing, then $v(x,y,z,) > 0$, that is $E_6\,(x_3, y_3, z_3)$ is globally asymptotically stable.

Remark 3.3.1

By $f(x,y)$ to be non decreasing [non - increasing] we mean;

If $x_1 < x_2, y_1 < y_2$ then $f(x_1, y_1) \le f(x_2, y)$ [$f(x_1, y_1) \ge f(x_2, y_2 +)$]

Thus, we have proved the following theorem.

Theorem 3.3.1:

Assuming that (H1), (H2), (H3), (H4), (H5), (H6) hold for the system (3.3.1) and the functions $\frac{xf(x)}{q(x)} - \frac{yg(y)}{r(y)}, \frac{yp(x)}{r(y)}$ are non increasing with either $\frac{xf(x)}{q(x)}$ or $\frac{yg(y)}{r(y)}$ is strictly decreasing and $\frac{yp(x)}{q(x)}$ is nondecreasing. Then the equilibrium $E_6(x_3, y_3, z_3)$ is globally asymptotically stable.

Remark 3.3.2

We have shown our work for the region A, but the results hold for the region

$B_1 = \{(x,y,z): 0 < x < x^* < k_1, 0 < y < y^* < \gamma, 0 < z < z^* < \beta\}$ as well. In fact, if we assume that

$$(p(x)-p(x^*))(x-x^*) > 0, (q(x)-q(x^*))(x-x^*) > 0,\ x \neq x^*$$

and $(r(y) - r(y^*))(y-y^*) > 0, y \neq y^*$ the theorems 3.1.1, 3.2.1 and 3.3.3 hold in a neighbour hood of (x^*, y^*, z^*)

Remark 3.3.3:

If E_6 $(x,y\,z)$ (i = 1, 2, 3) are globally asymptotically stable for the systems (3.1.1), (3.2.1), and (3.3.1) respectively, then all the trajectories of these systems in the positive cone will spiral toward (x,y,z), that is, non existence of limit cycles.

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 11-20

EFFECTS OF HIGH FREQUENCY ELECTROMAGNETIC WAVES ON HUMAN SKIN

Vijay Kumar* and P.P. Pathak**

(Received 25-05-2007, Revised 20-07-2007)

ABSTRACT

The penetration of high frequency electromagnetic waves (EMW) into the skin of human body and its possible consequences are studied. The observations are perfectly theoretically verified which show that the high frequency EMW is more harmful for the skin of human body living nearby as compared to that from the low frequency transmission tower. The power is absorbed by the skin of body due to the induction of electric field. The absorbed power density increases with frequency, may result into temperature rise in the skin of the body.

Key words : Electromagnetic waves, dermis, epidermis, penetrated electric field, absorbed power density.

INTRODUCTION

Our atmosphere contains electric and magnetic fields as well as electromagnetic fields (EMF). Cellular phones and transmission towers of these phones also produce EMF. All biological systems have complex electrical structure. Therefore some kind of interaction is eminent between the two. Huge amount of work has been done on this interaction in last decade or two. Giving main features of work so far along with the physical basis for them, here we have calculated penetrated electric fields and absorbed power at different depths of skin leading to different deseases.

It is well known that the control of physical processes and information transformation from one part of the body to another takes place through electrical signals. Nerve cells propagate electrical signals from sensors of pressures, temperature, light, sound etc. to the brain and return control signals to muscles and other tissue. Yet, if we try to influence these processes with external electrical inputs, we have relatively limited understanding of how a given electrical signal will affect various biological organs. What the safe limits of exposure are and how electrical signals are carried across the cell membranes or propagated along nerves.

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*Department of Physics, S.D. Bansal College of Technology, Indore (M.P.) India.
**Department of Physics, Gurukula Kangri University, Haridwar, 249404, India

When human body is exposed to EMW the skin of the human body- absorbs it partly. Healthy skin consists of epidermis and dermis. The epidermis, a vascular top layer up to about a thickness of 0.15 mm consists of an upper most layer of tough dead cells and a thin layer of living epithelial cells, which contain melanin pigments responsible for color of skin. The dermis below this, which varies from 1 to 2 mm in thickness, is invariably profused with blood supply. The dominant pigment is the hemoglobin, which exists in various states of molecules. Green light of solar radiation is strongly absorbed by the hemoglobin in the blood-rich dermis. Longer wavelength of visible light can penetrate several millimeter into the skins possibly passing all the way through the dermis and into the underlying layers of connective tissues and fat. The electrical properties of skin are determined by corneum having thickness of the order of 15 μm. The capacitive-resistive element of skin has a thickness of the order of 2 μm. This layer provides the protective barrier between the body tissues and environment.

The number of wireless telephone subscribers in India has increased at an extremely high rate in about last ten years. The rapid adoption of wireless telephones, especially handheld mobile phones, has occurred amidst controversary over whether this technology poses a risk on human skin. Like other devices that transmit radio signals, mobile phone emits electromagnetic energy in radio frequency range. At high power levels, radio frequency energy can rapidly heat the skin tissue and cause damage to it, burns. Mobile phones operate at power levels well below the level at which such direct heating occurs. However, an issue that has been the subject of research and debate for several years is whether long-term exposure to low level radio frequency electromagnetic energy from mobile phones could cause other types of adverse health effects, such as cancer.

The wireless cellular phone receives and transmits high frequency electromagnetic waves. Thus a blanket of high frequency electromagnetic field surrounds cellular phone users. A lot of studies have been carried out with particular reference to harmful effects of radiations. Rai [12] gives an useful collection of the work in this area and concluded that the results of a project task conducted by him on the population living around the Radio/TV transmission towers of Varanasi, are hazardous and "many people are vigorously suffering from radio frequency induced health problems but wrongly attributed to chemical, water, air and noise pollution,..........". Validity of this statistical survey may not be squarely doubtful because of distance dependence of occurrence of health problems. Radio frequency fields produce thermal effect in the skin of the body because of absorption of radiation. Thermoregulatory behavior is changed due to presence of microwave radiation [1]. Freund et al. [4,5] found that the use of cellular phones is harmful as the microwaves emitted by them affect the human slow brain potentials adversely. Many epidemiological studies have suggested that exposure to electromagnetic fields increased the risk of leaukemia and brain turnours [3,14-17]. Recently

Kawne et al. [6,7] Kumar et al. [8,9] Makar [11] and Logani [10] have also found the possibility of childhood leukemia/cancer risk due to field produced by transmission towers of TV, radio & mobile telephony and by transmission line.

Physics of the interaction of electric fields with human skin :

Biological system consists of complex physical subsystems. In order to understand them one has to start at most elementary level. Simplest level is effect of electric fields on biological fluids. These fluids contain a large number of components, including ions, polar molecules and colloidal particles. Current flow in these fluids is given by the sum of the drift and diffusion currents for each component. At low current densities the system is linear. At moderate and high current densities, non-linearties are observed. Next level of complexity arises due to interaction of fields with membranes that behave like porous solids for fields applied perpendicularly to their surface, and like viscous liquids for fields in the plane of the membrane. The membranes being inhomogeneous their different portions are affected differently by perturbing fields. Additionally the membranes are involved in active chemical reactions that change their porosity to various ions selectively so that electrical potentials and chemical signals may change the membrane's conductivity by orders of magnitude. The presence of external electric field makes it more complex, as the interaction between biological fluids and membranes is affected by selective transport of ions or molecules through membranes. The build up of charged ion layers at the surface and the way new molecules are incorporated into membranes, or are bound to its surface, is affected. As a consequence the performance of the cell is affected, and in turn, performance of the body part concerned is affected. For example, a biasing electric current across a pace maker cell in the heart will change its firing rate and thus the pumping rate of the heart. Barnes [2] has treated the whole process mathematically.

MATERIAL AND METHODS

Any transmission tower transmits high frequency electromagnetic wave (EMW) and due to this high frequency electric field is propagated around the tower. If any person lives around the 100 m premises of 10 kW transmiter, he is surrounded by the electric blanket of 7.746 V/m [12]. This high frequency electric field E penetrates inside the human body and affects the different parts of the body by different way. First of all this electric field affects the skin of the body.

A healthy skin consists two layers dermis and epidermis. If any person is surrounded by the electric field of E_0, then electric field after penetration up to z depth inside the skin of human body becomes

$$E = E_0 \, e^{(-z/\delta)} \tag{1}$$

where δ is skin depth of the body material as given by Pathak et al. [12]. Due to the induction of electric field E in dermis and epidermis of skin, a voltage is dropped across every cell of the skin. This voltage drop represents that there is absorption of power.

The voltage drop across the cell having thickness l is given by [12].

$$V = El \tag{2}$$

Due to the penetration of high frequency electromagnetic field, the voltage is dropped across each cell of skin, which represents that there is a absorption of power of radiation. The absorbed power density P by the layers of skin is given by

$$P = \sigma E^2 \tag{3}$$

where σ is conductivity of skin and E is induced electric field inside the skin.

RESULTS AND DISCUSSION

After penetration of high frequency electromagnetic wave (EMW) in different layers of human skin, the induced electric field in dermis, epidermis, skin having capacitive and resistive (C-R) element, skin having electrical properties are shown in Tables 1 to 3. The data of electric field in Tables 1, 2 and 3 are due to the radiation from tower of 10 kW which radiate the frequencies 10 GHz, 5 GHz and 2.45 GHz respectively. First column of Table 1,2 and 3 represents the induced electric fields inside the skin having C-R element due to the incident of high frequency EMW, which decreases rapidly as the distance of the subject from the tower increases. Second column of these three tables show the induced electric field inside the layer of skin having electrical property. This induced electric field decreases nearly uniformly with in the skin having C-R element as the distance from the tower is increased. Third column of above-mentioned tables represents the induced, electric field in the layer of skin epidermis. The electric field decreases more rapidly in this layer in comparison to previous layers as the distance increases from the tower. The fourth and fifth column of these tables show the induced electric field in the layer of skin dermis. The thickness of this layer is varied from 1 mm to 2 mm. The induced electric field in this layer is much less than previous layer. The induced electric field is lesser in layer of thickness 2mm than thickness of 1 mm. In all these tables the distances of the subject are taken from l m to 100 m from the transmission tower.

These three tables also show that the highest electric field is induced in the layer of skin having C-R element and lowest electric field is induced in the dermis layer of skin for any one particular frequency of radiation. These tables also show that the induced electric fields are higher for higher frequency of radiation.

Tables 4 to 6 reveal that the variations of absorbed power density of radiation by the skin having C-R element, skin having electrical property, epidermis and dermis due to the radiation of 10 MHz, 5 MHz and 2.45 MHz are faster than the variation of induced electric field in these layers respectively as power is proportional to square of the field. Absorbed power density is maximum for the layer of skin having C-R element for any particular frequency of the radiation and it becomes high as the frequency of radiation becomes high and lowest for the dermis layers of skin.

According to Tables 2 & 3, when the frequency of incident radiation increases from 2.45 GHz to 5 GHz, the induced electric field in different layers of skin decreases by 0.01%, 0.116%, 1.01%, 6.5% and 12.7% in skin having C-R element, skin having electrical property, epidermis, dermis layer of thickness 1mm and 2 mm respectively at a distance of 100 m from the transmission tower. The penetrated electric field inside these layers of skin decreases by 0.0026%,0.25%,2.4%, 15.72% and 28.1% respectively, at 100 m distance from the transmission tower when frequency of radiation increases 5 GHz to 10 GHz (Tables 1 &2).

Due to the induction of electric field, the power of electromagnetic wave (EMW) is absorbed inside the different layers of skin. The absorbed power density is increased as the frequency of incident radiation is increased. According to Tables 5 and 6, as the frequency of radiation increases from 2.45 GHz to 5 GHz, the absorbed power density increases by 43.61 %, 43.46%, 42.47%, 35.7% and 26.5% respectively in skin layer having C-R element, skin layer having electrical property, epidermis layer of skin, dermis layer of skin of thickness 1mm and of thickness 2 mm at a distance 100 m from the transmission tower. Tables 4 and 5 show this increase in absorbed power density as 61.9%, 61.7%, 60.14%, 47.09% and 26.3% respectively, at 100 m distance from the transmission tower, when the frequency of incident radiation increases 5 GHz to10 GHz.

Thus Tables 4 to 6 show that as the frequency of radiation increases, the absorbed power density in skin increases very fast and the absorption of power decreases as the distance from the tower increases. The absorption of radiation can increase the temperature of the skin.This is significant since the, thermal receptors of the nervous system are located there and will indicate pain when the incident power density reaches levels that could thermally damage the tissue. With surface cooling of the skin, however, by natural environmental conditions or by controlled clinical procedures, the temperature increases may be higher in the fat or muscle. The peak absorption in the various tissues may vary a wide range with fat thickness and frequency.

CONCLUSION

The above analysis represents that the high frequency electromagnetic field is induced inside the different layers of the skin and these layers of skin absorb power, which is harmful for the tissues of the skin. The absorption of power of high frequency electromagnetic field by the skin having C-R element is highest which is harmful for the human body because this layer provides the protective barrier between the human skin tissues and environment. Thus the tower which radiate high frequency electromagnetic waves should be located away from the thickly populated area.

Table 1: Penetrated electric field in different layers of skin when 10 GHz EMW is incident on the human body

Distance from the tower (m)	Penetrated electric field (V/m) in				
	Skin having CR element (2 μm)	Skin having electrical property (15 μm)	Epidermis (0.15 mm)	Dermis (1 mm)	Dermi (2 mm
1	774.13	771.19	741.99	578.7	432.3
10	77.413	77.119	74.19	57.87	43.23
20	38.7	38.55	37.09	28.93	21.61
30	25.8	25.7	24.73	19.29	14.41
40	19.35	19.27	18.54	14.46	10.8
50	15.48	15.42	14.84	11.57	8;64
60	12.9	12.85	12.36	9.64	7.205
70	11.05	11.01	10.6	8.266	6.175
80	9.674	9.63	9.27	7.23	5.4
90	8.59	8.56	8.23	6.42	4 799
100	7.74	7.711	7.419	5.78	4.32

Table 2 : Penetrated electric field in different layers of skin when 5 GHz EMW is incident on the human body

Distance from the tower (m)	Penetrated electric field (V/m) in				
	Skin having CR element (2 μm)	Skin having electrical property (15 μm)	Epidermis (0. 15 mm)	Dermis (1 mm)	Dermis (2 mm)
1	774.37	773.05	759.96	682.26	600.93
10	77.43	77.3	75.99	68.22	60.09
20	38.71	38.65	37.99	34.11	30.04
30	25.81	25.7	25.33	22.74	20.03
40	19.359	19.32	18.99	17.03	15.023
50	15.487	15.46	15.19	13.64	12.018
60	12.9	12.88	12.66	11.37	10.015
70	11.06	11.04	10.85	9.74	8.584
80	9.670	9.66	9.49	8.52	7.511
90	8.590	8.58	8.43	7.58	6.676
100	7.74	7.73	7.599	6.82	6.009

Table 3: Penetrated electric field in different layers of skin when 2.45 GHz EMW is incident on the human body

Distance from the tower (m)	Penetrated electric field (V/m) in				
	Skin having CR element (2 μm)	Skin having electrical property (15 μm)	Epidermis (0. 15 mm)	Dermis (I mm)	Dermis (2 mm)
1	774.45	773.9'	767.78	730.29	688.61
10	77.44	77.39	76.77	73.029	68.86
20.	38.72	38.69	38.38	36.51	34.43
30	25.81	25.79	25.59	24.34	22.953
40	19.36	19.34	19.19	18.257	17.215
50	15.488	15.47	15.35	14.605	13.772
60	12.9	12.89	12.79	12.17	11.476
70	11.062	11.05	10.96	10.432	9.836
80	9.678	9.67	9.59	9.126	8.605
90	8.598	8.59	8.52	8.108	7.645
100	7.74	7.73	7.677	7.302	6.886

Table 4: Absorbed power density by different layers of skin when 10 GHz EMW is incident on the human body

Distance from the tower (m)	Absorbed power density (V/m) by				
	Skin having CR element (2 μm)	Skin having electrical property (15 μm)	Epidermis (0.15 mm)	Dermis (I mm)	Dermis (2 mm)
1	6172.55	6125.76	5670.65	3449.4	1924.89
10	61. 72	61.25	56.7	34.49	19.24
20	15.42	15.3	14.16	8.62	4.8
30	6.85	6.80	6.29	3.83	2.13
40	3.85	3.82	3.54	2.15	1.2
50	2.468	2.449	2.268	1.378	0.768
60'	1.714	1.7	1.575	0.957	0.534
70	1.26	1.257	1.157	0.703	0.392
80	0.963	0.955	0.385	O.538	0.3
90	0.76	0.754	0.697	0.424	0.237
100	0.617	0.612	0.567	0.344	0.192

Table 5: Absorbed power density by different layers of skin when 5 GHz EMW is incident on the human body

Distance from the tower (m)	Absorbed power density (kW/m^3) in				
	Skin having CR element (2 μm)	Skin having electrical property (15 μm)	Epidermis (0.15 mm)	Dermis (l mm)	**Dermis (2 mm)**
1	2350.62	2342.61	2263.95	1824.67	1415.58
10	23.5	23.42	22.63	18.246	14.155
20	5.876	5.855	5.657	4.56	3.538
30	2.61	2.603	2.515	2.027	1.572
40	1.469	1.463	1.413	1.137	0.884
50	0.94	0.936	0.904	0.7298	0.566
60'	0.652	0.65	0.628	0.5068	0.393
70	0.479	0.477	0.461	0.372	0.288
80	0.366	0.365	0.353	0.285	0.221
90	0.289	0.288	0.278	0.225	0.174
100	0.235	0.234	0.226	0.182	0.1415

Table 6: Absorbed power density by different layers of skin when 2.45 GHz EMW is incident on the human body

Distance from the tower (m)	Absorbed power density (kW/m^3) in				
	Skin having CR element (2 µm)	Skin having electrical property (15 µm)	Epidermis (0.15 mm)	Dermis (1mm)	Dermis (2 mm)
1	1325.49	1323.61	1302.76	1178.64	1047.94
10.	13.25	13.23	13.02	11.78	10.479
20	3.313	3.308	3.255	2.945	2.619
30	1.472	1.469	1.447	1.309	1.164
40	0.828	0.826	0.813	*0.736*	0.654
50	0.53	0.529	0.52	0.471	0.419
60	0.3677	0.3676	0.361	0.327	0.291
70	0.27	0.27	0.265	0.24	0.213
80	0.2069	0.2067	0.203	0.184	0.163
90	0.1633	0.163	0.16	0.145	0.129
100	0.1325	0.1323	0.13	0.117	0.104

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 21-26

ON CHARACTERIZATION OF SOME SEQUENCES

Geeta Bhakta Joshi*, Shailendra Kumar Mishra**
and Shankar Raj Pant***

(Received 03-08-2007)

ABSTRACT

Let t be a positive number sequence and let $\Omega(t) = \{x = (x_k): x_k = 0(t_k)\}$. This space has been introduced by Fricke and Fridy [1]. The main objective of this paper is to characterize the classes $(\ell_\infty(\Delta), \Omega(t)), (c_0(\Delta), \Omega(t))$ and $(c(\Delta), \Omega(t))$.

INTRODUCTION

Let ω denote the space of all complex sequences $\{x_k\}_{k=1}^{\infty}$. Through this paper, unless or otherwise specified $p > 0$ will denote a constant real number. The following classes of sequences [2,6] will be important in our discussion:

$$\ell_\infty = \{x = (x_k) \in \omega: \sup_k |x_k| < \infty\}$$

$$c = \{x = (x_k) \in \omega: |x_k - \ell| \to 0 (k \to \infty) \text{for some } \ell \in C\}$$

$$c_0 = \{x = (x_k) \in \omega: |x_k| \to 0 (k \to \infty)\}$$

If $\Delta x = x_{k+1} - x_k$, then

$$\ell_\infty(\Delta) = \{x = (x_k): \Delta x \in \ell_\infty\}$$

$$c(\Delta) = \{x = (x_k): \Delta x \in c\}$$

$$c_0(\Delta) = \{x = (x_k): \Delta x \in c_0\}$$

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Let λ be an arbitrary nonempty subset of ω. We define,

$$\lambda^\beta = \left\{a = (a_k) \in \omega: \sum_{k=1}^{\infty} a_k x_k \text{converges for each } x \in \lambda\right\},$$

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*Department of Mathematics, pulchowk campus / Institute of Engineering, Tribhuvan University, Lalitpur, Nepal.
**Department of Mathematics, pulchowk campus / Institute of Engineering, Tribhuvan University, Lalitpur, Nepal.
***Ex-Chairman, Central Department of Mathematics, Tribhuvan University, Kathmandu, Nepal.

where, λ^{β} is called β⁻(or generalized Köthe- Toeplitz dual of λ). We determine β - duals of $c_0(\Delta)$ and $c(\Delta)$.

Theorem 2.1:

Let $(p_k) \in \ell_{\infty}$ Then $(c_0(\Delta))^{\beta} = \bigcup_{n>1}\left\{a=(a_k)\in\omega: \sum_{k=1}^{\infty} a_k k_n^{-\frac{1}{p_k}} \text{converges and} \sum_{k=1}^{\infty}|R_k|n^{-\frac{1}{p_k}} < \infty\right\}$

where $R_k = \sum_{r=k}^{\infty} a_r = D_0(p)$

Proof:

Let $a \in D_0(p)$ and $x \in c_0(\Delta)$. Then there exists a positive integer $n > 1$ such that $|\Delta x_k|^p < \frac{1}{n}$ for sufficiently large value of k. we have.

$$\sum_{k=1}^{m} a_k x_k = \sum_{k=1}^{m} R_k \Delta x_k - R_{m+1}\sum_{k=1}^{m}\Delta x_k \,(m \in N)$$

As $\sum_{k=1}^{\infty}|R_k||\Delta x_k| \le \sum_{k=1}^{\infty}|R_k|n^{-\frac{1}{p_k}} < \infty$, it follows that it is absolutely convergent. Observe, the convergence of $\sum_{k=1}^{\infty} a_k K_n^{\frac{-1}{p}}$ implies that $R_{m+1}\sum_{k=1}^{m} n^{-\frac{1}{p_k}} = o(1)$. Hence $\sum_{k=1}^{\infty} a_k x_k$ converges for each $x \in c_0(\Delta)$. This implies that $a \in (c_0(\Delta))^{\beta}$. Next $a \in (c_0(\Delta))^{\beta}$, then for any $x \in c_0(\Delta)$, $\sum_{k=1}^{\infty} a_k x_k$ converges. Define a sequence $y = y_k$ by $y_k = kn^{-\frac{1}{p_k}}$. Then it is clear that $y \in c_0(\Delta)$ *and* $\sum_{k=1}^{\infty} a_k k_n^{-\frac{1}{p_k}}$ converges. Suppose that $a \in (c_0(\Delta))^{\beta}$, but for some $n > 1$, $\sum_{k=1}^{\infty}|R_k|n^{-\frac{1}{p_k}} = \infty$. It follows that $R = (R_k)$ does not belong to $(c_0(\Delta))^{\beta}$. Then there is a sequence

$u=\left(\frac{1}{k}\right)^{\infty} \in c_o(\Delta)$ such that $\sum_{k=1}^{\infty} \frac{R_k}{k}$ is not convergent. Although, if we define a sequence

$$z = z_k \text{ by } z_k = \sum_{m=1}^{k} \frac{1}{m}, \text{ then } z \in c_0(\Delta) \text{ and}$$

$\sum_{k=1}^{\infty} a_k z_k = \sum_{k=1}^{\infty} a_k \left(\sum_{m=1}^{k} \frac{1}{m} \right) = \sum_{k=1}^{\infty} \frac{R_k}{k} \neq O(1)$. *Hence* $\sum_{k=1}^{\infty} a_k z_k$ is not convergent. This contradicts the fact that $a \in (c_0(\Delta))^{\beta}$. Hence we must have $\sum_{k=1}^{\infty} |R_k| n^{-\frac{1}{p_k}} < \infty$.

Theorem 2.2:

$$(c(\Delta))^{\beta} = (c_0(\Delta))^{\beta} \cap \left\{ a = (a_k) \in \omega : \sum_{k=1}^{\infty} k a_k \text{converges} \right\}$$

Proof:

Not that for any two sequences S and T, we have $(S \oplus T) = S^{\beta} \cap T^{\beta}$.

As $\quad c(\Delta) = c_0(\Delta) \oplus \{(k)\}$,

therefore,

$$(c(\Delta))^{\beta} = (c_0(\Delta))^{\beta} \cap \{(k)\}^{\beta} = (c_0(\Delta))^{\beta} \cap \left\{ a = (a_k) \in \omega : \sum_{k=1}^{\infty} k |a_k| < \infty \right\}$$

Now,

$|a_k| \leq |a_k| n^{\frac{1}{p}} k n^{-\frac{1}{p}}$ for each $k \in n$, it follows that $\sum_{k=1}^{\infty} |a_k| < \infty$.

Thus, we find that

$$(c_0(\Delta))^{\beta} = (c_0(\Delta))^{\beta} \cap \left\{ a = (a_k) \in \omega : \sum_{k=1}^{\infty} k a_k \textit{ converges} \right\}.$$

MATRIX TRANSFORMATION

Fricke and Fridy [1] defined the following sequence space: Let $t=(t_k)$ be a positive sequence, then $\Omega(t)=\{x=(x_k): x_k = 0(t_k)\}$. Note that if t is a nonzero constant sequence, then $\Omega(t)=\ell_\infty$. Let X and Y be any two nonempty subsets of ω and $A=(a_{nk})_{n,k}$ be an infinite matrix of complex numbers. For every $x=(x_k)\in X$, the sequence $A_n(x)=AX$ where $A_n(x)=\sum_{k=1}^{\infty} a_{nk}x_k$. The sequence AX is called the transformation of X by the matrix A. By (X, Y) we mean the class of matrices A which transform X into Y. The aim of this section is to characterize the classes $(\ell_\infty(\Delta), \Omega(t)), (c_0(\Delta), \Omega(t))$ *and* $(c(\Delta), \Omega(t))$

Theorem 3.1:

$A\in(\ell_\infty(\Delta),\ \Omega(t))$ if and only if

(i) $\left(A_n\left(kN^{\frac{1}{p}}\right)\right)_n \in c, N>1$

(ii) $R\in(\ell_\infty,\Omega(t))$ where $R=(r_{nk})=\left(\sum_{r=k}^{\infty} a_{nr}\right)$

Proof:

If $A\in(\ell_\infty(\Delta),\Omega(t))$, then the series $\sum_{k=1}^{\infty} a_{nk}x_k$ is convergent and $(A_n(x))_n \in\Omega(t)$ For each n and $x\in\ell_\infty(\Delta)$. As $x=\left(kN^{\frac{1}{p}}\right)_k \in\ell_\infty(\Delta)$, it follows that $\sum_{k=1}^{\infty} a_{nk}\left(kN^{\frac{1}{p}}\right)$ converges for each n. Therefore (i) is necessary. To see, the condition (ii) is necessary. Let us assume that (ii) is false. Then there is a sequence $x=(x_k)\in\ell_\infty$ with $\sup_r |x_r|=1$ *such that* $\sum_{r=1}^{\infty} r_{nr}x_r \neq O(1)$. We now define a sequence $y=(y_r)$ by $y_r=\sum_{k=1}^{r} x_k$. Then $y\in\ell_\infty(\Delta)$ and

$\sum_{r=1}^{\infty} a_{nr} y_r = \sum_{r=1}^{\infty} r_{nr} x_r \neq O(1)$. This contradicts that $A \in (\ell_\infty(\Delta), \Omega(t))$. Thus the condition (ii) is necessary.

To prove the sufficiency part of the theorem, assume that the given conditions of the theorem are satisfied. Let $x \in \ell_\infty(\Delta)$. Then there is an integer $N > \max\left(1, \sup|x_k|^p\right)$. We write,

$$A_n(m,x) = \sum_{k=1}^{m} a_{nk} x_k = \sum_{k=1}^{m} r_{nk} \Delta x_k - r_{n+1} \sum_{k=1}^{m} \Delta x_k \,(m \in N),$$

since, $\sum_{k=1}^{\infty} |r_{nk}| |\Delta x_k| \le \sum_{k=1}^{\infty} |r_{nk}| N^{\frac{1}{p}}$ and $\left(\sum_{k=1}^{\infty} |r_{nk}| N^{\frac{1}{p}} \right) \in \Omega(t)$. Therefore by corollary 2 in [2], the convergence of $\sum_{k=1}^{\infty} a_{nk} \left(\sum_{i=1}^{k} N^{\frac{1}{p_i}} \right)$ implies that

$$r_{n+1,m} \sum_{k=1}^{m} N^{\frac{1}{p_k}} = O(1)$$

Hence, $A_n(x) = \sum_{k=1}^{\infty} a_{nk} x_k = \sum_{k=1}^{\infty} r_{nk} \Delta x_k$, since $x \in \ell_\infty(\Delta)$ if and only if $\Delta(x) \in \ell_\infty$. Therefore, by condition (ii) it follows that $A_n(x)$ exists for each $x \in \ell_\infty(\Delta)$

and $\qquad AX = (A_n(x))_n \in \Omega(t).$

Thus, $\qquad A \in (\ell_\infty(\Delta), \Omega(t)).$

Theorem 3.2 :

$A \in (c_0(\Delta), \Omega(t))$ if and only if

(i) $\left\{ A_n\left(kN^{\frac{-1}{p}}\right) \right\}_n \in c, N > 1$

(ii) $R \in (c_0, \Omega(t))$

The proof of this theorem follows immediately from the arguments given in Theorem 3.1 and Theorem 2.1.

Theorem 3.3:

$A \in (c(\Delta), \Omega(t))$ if and only if

(i) $A \in (c_0(\Delta), \Omega(t))$

(ii) $\left(\sum_{k=1}^{\infty} k a_{nk}\right)_n \in \Omega(t)$

The proof of this theorem follows immediately from Theorem 3.2 and Theorem 2.2.

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 27-35

SPECTROPHOTOMETRIC DETERMINATION OF Ir(III) WITH CYSTINE IN ALKALINE MEDIUM#

Anjali Goel*, Savita Devi* and Shailja*

(Received 09-02-2007, Revised 25-08-2007)

ABSTRACT

A method for the determination of Ir (III) with the cystine is described. The method is based on the Ir (III) catalysed oxidation of cystine by hexacyanoferrate (III) in aqueous alkaline medium. The method is used in the range of 0.643 to 6.43 μgcm^{-3}. The molar absorptivity and sandell's sensitivity are found $2.93x10^3$ to 4.2×10^3 $dm^3 mol^{-1} cm^{-1}$ and 0.046 to 0.0066 μgcm^{-2} respectively. The value of correlation coefficient lies between - (-) 0.9992 to 0.9961. The effect of few interferrants has also been worked out. For developing the calibration curves in terms of absorbance or initial rates or pseudo first order rate constant vs. [Ir (III)] plots, absorbance was noted at λ_{max} of 420 nm. The results show that the present method is more simple and sensitive than the reported methods and especially useful in absence of the expected interferrants. The standard deviation estimated from six independent determination in sample of Ir (III) for the determination in sample of Ir (III) for the concentration of 6.7×10^{-6} M are 0.0845 to 0.2200.

Keywords.-Microgram determination, Ir (III), Hexacyanoferrate (III), Oxidation, Cystine

INTRODUCTION

Iridium, a metal of the platinum family, is white, similar to platinum, but with a slight yellowish cast. It is very hard and brittle making it very hard machine, form, or work. Due to their corrosion resistant nature, iridium alloys have found a wide range of applications in both the chemical industry and manufacture. Iridium is also a minor constituent of most platinum metal deposits. Therefore, high selective, sensitive, rapid and economical methods are needed for its trace determination. Neutron activation analysis [6] atomic absorption spectrometry [4] or inductively coupled plasma atomic emission spectrometry [7] may be used for this purpose. However the necessary instruments are expensive, day to day maintenance costs are high and the methods are not free from various types of interferences.

Thus in the present study an attempt has been made to determine iridium (III) concentration in microelements in aqueous alkaline medium by kinetic spectrophotometric

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#This paper was presented at 43rd Annual Convention of Chemists organised by Indian Chemical Society at Aurangabad.

*Department of Chemistry, Kanya Gurukula Mahavidyalaya, Gurukula Kangri Vishwavidyalaya, Haridwar

method. The method is based on the oxidation of cystine by hexacyanoferrate (III), catalyzed by iridium (III) in aqueous alkaline medium.

EXPERIMENTAL

All chemicals used were of Analar grade. Hexacyanoferrate (III) was used after recrystallisation. All the solutions and reaction mixture were prepared by using double distilled water. Absorbance was recorded on Sys. UV-Visible spectrophotometer- 117. λ_{max} for the reaction mixture was observed to be 420 nm. $IrCl_3.10H_2O$ (SRL) was prepared by dissolving the sample in dil HCl. In order to optimize the variable, effect of concentration of each reagent on the rate of reaction was studied. Following were the finally worked out conditions for running the kinetic sets for the purpose of determination of Ir (III) in aqueous alkaline medium.

[Cystine] = 3×10^{-3} mol dm^{-3}; [HCF(III)] = 3×10^{-4} mol dm^{-3}; [OH^-] = 4×10^{-1} mol dm^{-3} $\mu = 0.7$ mol dm^{-3}, λ_{max} 420 nm; Temp. = 35+0.1°C.

Following procedure was adopted for preparation of the calibration curves. A definite volume of stock solution of hexacyanoferrate (III) was mixed with calculated volume of stock solution of Ir (III), sodium hydroxide, potassium chloride and water and stirred a little with the help of the pipette. The mixture and stock solution of cystine was then clamped in a thermostat at 35+0.1°C. After 30 min., a required amount of cystine solution was added to the mixture and stirred to start the reaction. All the additions were made in amounts calculated for maintaining the concentration of different reagents as mentioned above. Different sets were prepared in a similar manner varying the Ir (III) concentration. Aliquots were withdrawn from the reaction mixture after repeated intervals of 5 min. and the absorbance was recorded. The absorbance of reaction mixture goes on decreasing with the passage of time. The absorbance vs. time plots were made for all the sets. Initial rates (dA/dt) were evaluated after 5 min. from the start of the reaction by using plane mirror method. The pseudo first order rate constant (k_1) were calculated by Guggenheim's method.

RESULTS AND DISCUSSION

The present study deals with kinetic spectrophotometric studies on the microgram determination of iridium (III), while it catalyses the cystine and hexacyanoferrate (III) redox system in aqueous alkaline medium. The reaction was followed by noting the absorbance of the reaction mixture after time 5, 10, 15, 20 min. from the start of the reaction. Linear calibration curve were obtained in terms of type 'A', type 'B', type 'C', type 'D', type 'E' and type 'F' i.e. A_5 or A_{10}, or A_{15} or A_{20} or [$(dA/dt)_5$] or k_1 vs. [Ir (III)] plots respectively Fig. (1-3).

The stoichiometry of the reaction was studied by estimating the amount of hexacyanoferrate (II) ions produced after definite interval of time with standard solution of ceric (IV) sulphate using ferroin as redox indicator. The studies show that four moles of hexacyanoferrate (III) are used for the oxidation of one mole of cystine. Based on the experimental results, it can be proposed that reaction propceeds through complex formation between anion of cystine and iridium (III) which slowly disproportionate into Ir (I) and intermediate product. Ir (I) reoxidised by two moles of hexacyanoferrate (III) ions to Ir (III) through electron transfer mechanism and the intermediate product decomposes to final product.

STATISTICAL INTERPRETAION

The results presented in table -1 show that Beer's law was obeyed in the range of 0.643-6.44 μgcm^{-3} of iridium (III). The value of slope of the calibration curves, molar absorptivity and sandell's sensitivity indicated that the sensitivity of the method is good. Molar absorptivity values are 2.93×10^3 to 4.2×10^3 dm^3 mol^{-1} cm^{-1}. Sandell sensitivity ranges from 0.046 to 0.066 μgcm^{-2}. The value of slope lies between 2.89×10^{-3} to 0.1696×10^{-3}. The intercept and correlation coefficient are also calculated. The high value of correlation coefficient (r) and low value of interecpt on the ordinate validates the linearity of the calibrations graphs. The standard deviations, evaluated from six determinations in sample of 0.643 to 6.44 μgcm^{-3} of iridium (III), were 0.08485 to 0.22 are considerably low. These are good for the trace determination of Ir (III). The correlation coefficient (r) -0.9992 to - 0.9961 and coefficient of determination (r^2) - 0.9922 to 0.9985 indicates high precision involved in the determination and good correlation of the data. Percentage recovery on the basis of six determinations is 98.0 to 96.8%.

INTEREFERENCE EFFECT OF CATIOS AND ANIONS

The effect of various ions on the determination of Ir (III) by this method has been studied. It has been observed that the presence of cations- Na^+, K^+, NH_4^+, As^{+++}, Mn^{++}, Ca^{++}, Cr^{+++}, Li^{++}, Sr^+ and anions- carbonate, acetate, nitrate, thiocyanate, EDTA, oxide, oxalate, iodate, molybdate, sulphate do not interfere in the estimation of Ir (III). The other metal ions of the same nature must be precipitated before undertaking the proposed method[8].

COMPARSION WITH OTHER METHODS

The result of present method are compared with other reported spectrophotometric methods in Table-2. From the data it can be revealed that the reported methods are time consuming, occurs at very high temperature and in the organic solvents like chloroform, ether etc. As compared with these methods, the present method is considerably less complicated, occurs at room temperature and in aqueous medium. The method is also sensitive and require low reagent concentration. The linear range of detection for the present method is quite good i.e. 0.643 to 6.44 μgcm^{-1} . The absorptivity value ranges from 2.93×10^3 to 4.2×10^3 dm^3

Table - 1

Characterstics of various types of calibration curves for the proposed method :

S.No.	Parameters	'A' plot	'B' plot	'C' plot	'D' plot	'E' plot	'F' plot
1.	Beer's Limit (μg ml^{-1})	0.643-6.44	0.643-6.44	0.643-6.44	0.643-6.44	0.643-6.44	0.643-6.44
2.	Molar absorptivity (dm^3 mol^{-1} cm^{-1})×10^{-3}	2.93	3.23	3.63	4.20	—	—
3.	Sandell's Sensitivitiy (μgcm^{-2})	0.0655	0.0594	0.0529	0.0458	—	—
4.	Correlation coefficient (r)	(-)0.9981	(-) 0.9961	(-)0.9980	(-) 0.9992	(-) 0.9986	(-)0.9984
5.	Coefficent of determination (r^2)	0.9962	0.9922	0.9961	0.9985	0.9972	0.9969
6.	Intercept (from regression equation)	0.2328	0.3387	0.3361	0.3347	0.0001	0.00005
7.	Slope (From regression equation)	(-) 0.0029	(-) 0.0034	(-) 0.0038	(-) 0.0044	0.000046	0.00017
8.	Standard deviation	0.0849	0.06928	0.09798	0.05657	0.16	0.22
9.	% Recovery	98.0	98.2	97.6	98.8	97.7	96.8

Table - 2

Comparison of the present method for iridium determination with some of the reported methods.

Reagent	Medium	Heating	Molar absorptivity dm³ mol⁻¹ cm⁻¹	Linear Range µg cm⁻³	Reference
Difurfuryl-thiocarbohydrazone (DCE)	Ethyle acetate	No Heating	4.15×10^4	0.93-3.73 [lr(III)]	(1)
1-Phenyl-4, 4-6-trimethyl 1 (1H, 4H) - pyrimidine-2-thiolate (PTPT)	$CHCl_3$	60°C (5 min.)	3.38×10^3	3.8-42.0 [lr(III)]	(3)
2- Mercapto-4, methyl 5-phenylazopyrimidine (MFAP)	$CHCl_3$	No Heating	9.5×10^3	0.60-9.0	(2)
Bis (thiopheno-2-aldehydo) -thiocarbohydrazone (BTATCH)	Ethyle acetate	100°C (30 min.)	3.2×10^4	1.2-4.2 [lr(III)]	(5)
Present Method	Aqueous Alkaline	35°C	2.933×10^3 to 4.2×10^3	0.643-6.44 [lr(III)]	—

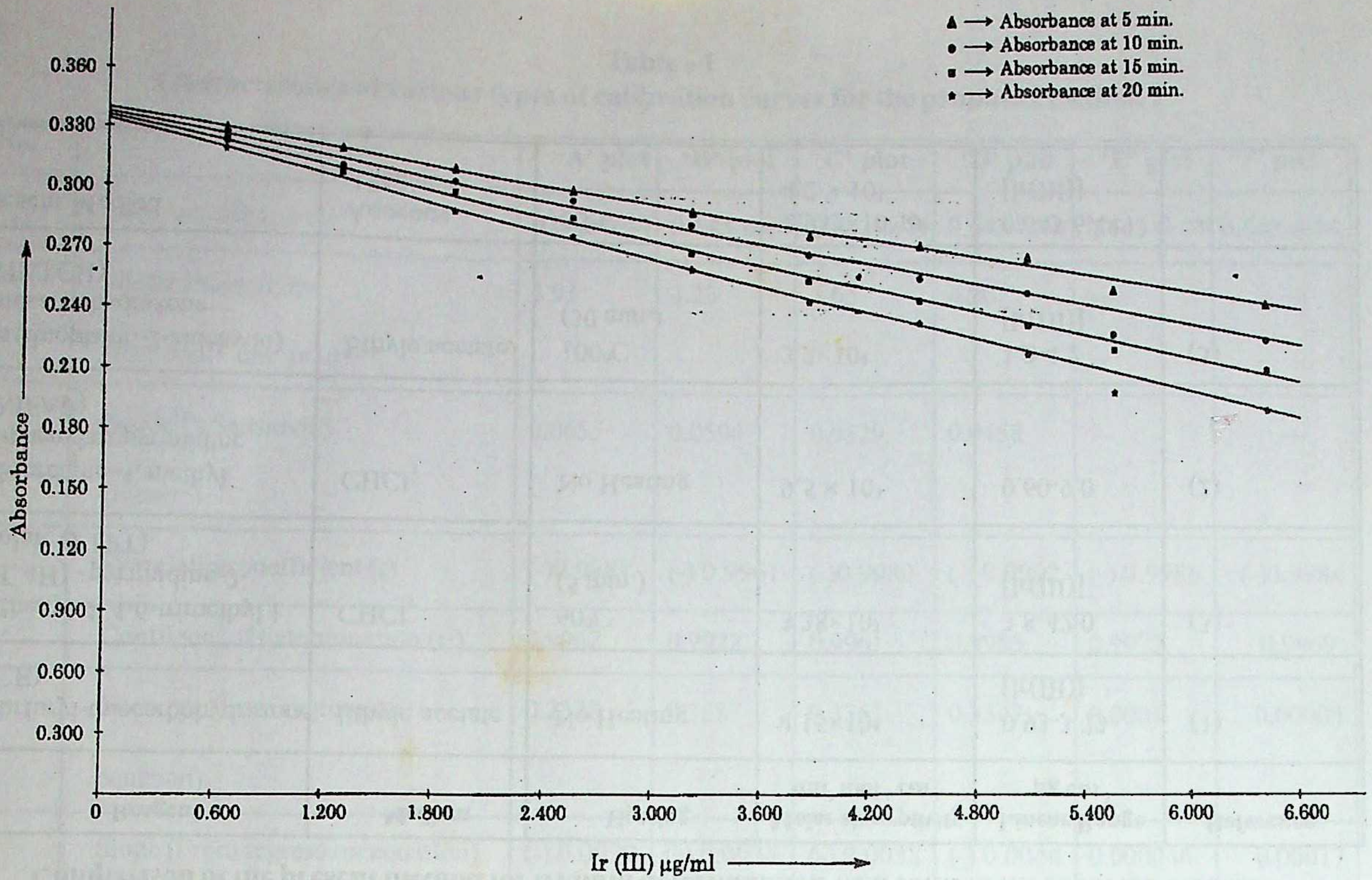

Fig. 1 : Plots between absorbance and [Ir(III)] in μg/ml. after time 5,10, 15 and 20 min. from the start of reaction.

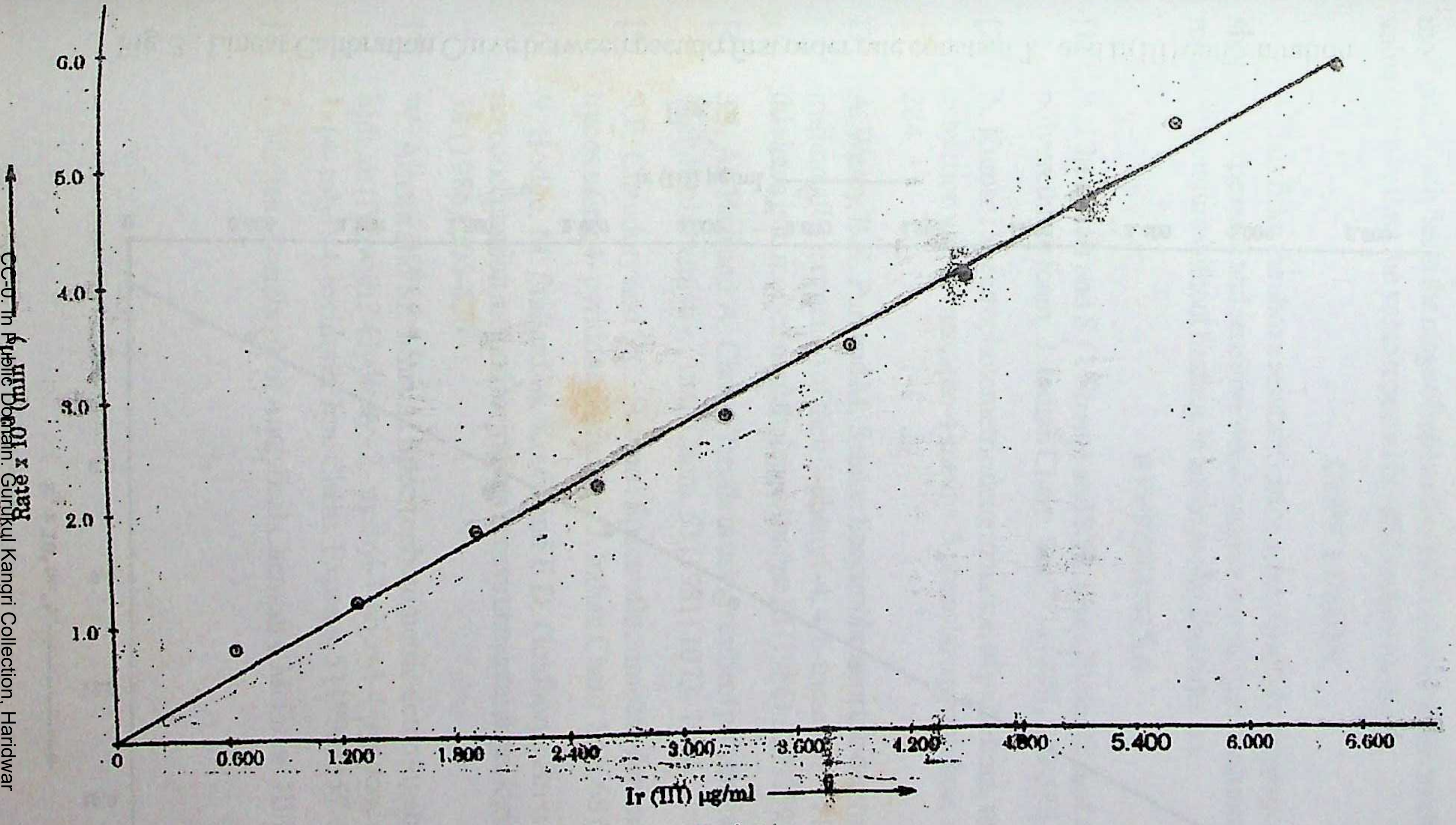

Fig. 2

Fig. 2 : Linear Calibration Curve between reaction rate and Ir(III) concentration.

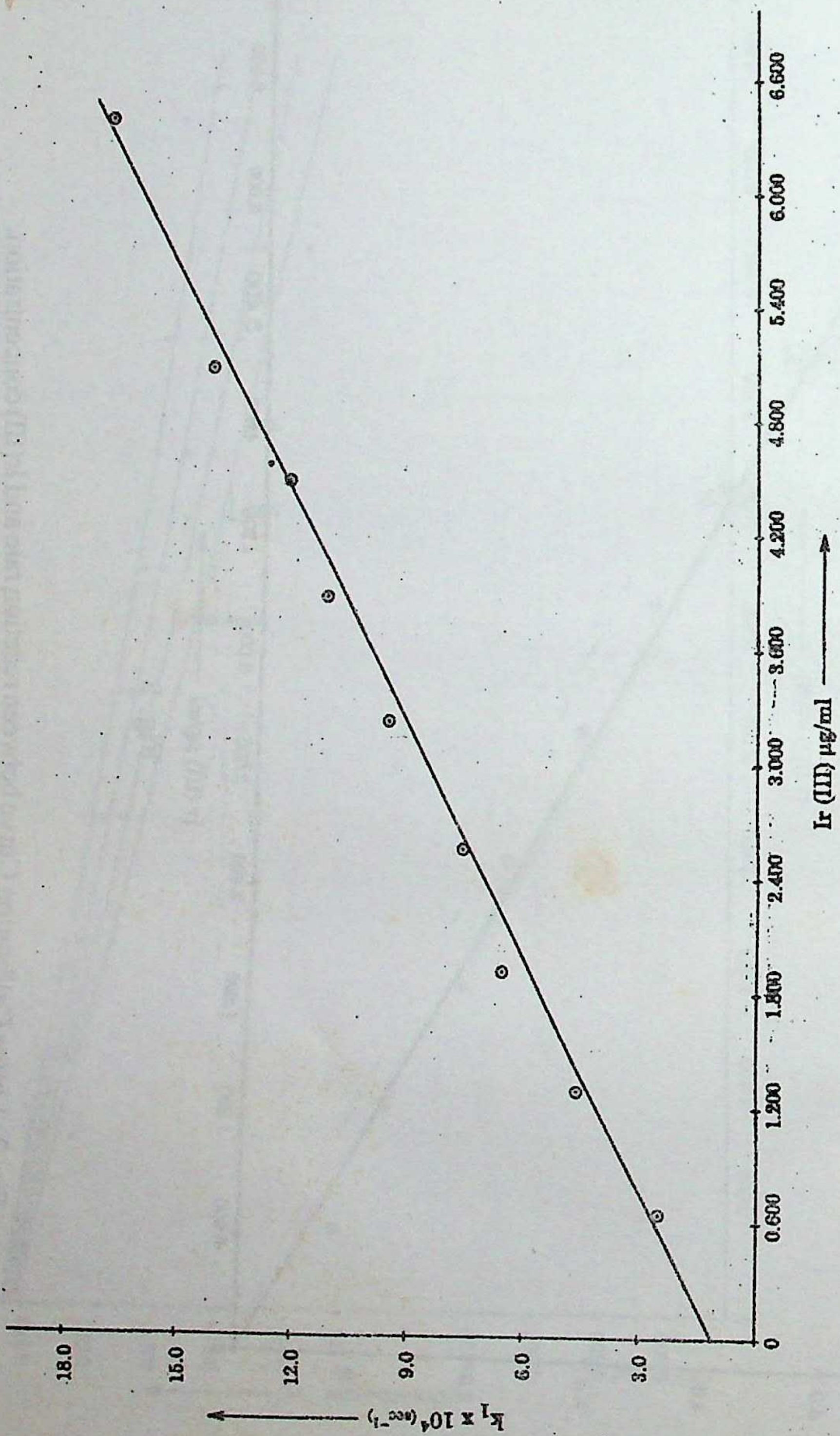

Fig. 3

Fig. 3 : Linear Calibration Curve between pseudo first order rate constant 'k', and Ir(III) concentration

mol^{-1} cm^{1} which lies in the range of reported methods in chloroform medium. But that absorptivity value is lower than the value reported in ethyl acetate medium.

CONCLUSION

Thus from the above results it can be concluded that the proposed method is relatively simple, inexpensive and sensitive which require low reagent concentration and can be used at room temperature without heating, in aqueous alkaline medium.

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 37-44

RELATED FIXED POINTS IN MENGER SPACES

B.D. Pant*, R.C. Dimri and N.S. Gariya****

(Received 30-04-2007, Revised 01-09-2007)

ABSTRACT

Here we prove some results connecting the fixed points of two mappings on two Menger spaces. We generalize some of the previous known results on related fixed points in two metric spaces.

Mathematics Subject Classifications (2000): 47H10, 54H25

Keywords : complete Menger space, fixed point.

INTRODUCTION

Menger [7] introduced the notion of probabilistic metric spaces (PM-spaces). Sehgal and Bharucha-Reid [13] initiated the study of fixed points in a subclass of probabilistic metric spaces. They extended the notion of contraction and local contractions to the setting of Menger spaces. The study of this space expanded rapidly with the pioneering work of Schweizer and Sklar [11].

Fisher [5, 6] has investigated the conditions for the existence of a relation connecting the fixed points of two mappings in two different metric spaces. Further generalizations of these results were obtained by Aliouche and Fisher [1, 2] and Namdeo and Fisher [8].

Pant [9] obtained probabilistic generalization of some results of Fisher [*op. cit.*] to two Menger spaces. Here we generalize and modify these results under a different contraction condition in two Menger spaces.

Now, we give some definitions and notations from earlier work[12].

A mapping $F: R \rightarrow R^+$ is said to be a distribution function if it is non-decreasing left-continuous with $\inf F = 0$ and $\sup F = 1$.

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*Department of Mathematics R.H.Govt.Postgraduate College, Kashipur (U.S. Nagar)
Uttarakhand 244713 - India
** Department of Mathematics, H.N.B. Garhwal University Post Box-100 Srinagar (Garhwal)
Uttarakhand-246174 - India E-mail: dimrirc@gmail.com

Definition 1:

A probabilistic metric space is an ordered pair (X, F), where X is an abstract st and F is a mapping of $X \times X$ into D i.e., associates a distribution function (p, q) with every pair (p, q) of points in X. We shall denote the distribution function (p, q) by $F p, q$. The functions Fp, q are assumed to satisfy following conditions:

(PM-1) $F_{p,q}(x) = 1$ for all $x > 0$ iff $p = q$,

(PM-2) $F_{p,q}(0) = 0$,

(PM-3) $F_{p,q} = F_{q,p}$,

(PM-4) If $F_{p,q}(x) = 1$ and $F_{q,r}(y) = 1$ then $F_{p,r}(x+y) = 1$.

Definition 2:

A triangular norm (briefly, a t - norm) is a function t: $[0, 1] \times [0, 1] \to [0, 1]$ satisfying the following conditions:

(i) $t(a, 1) = a$ and $t(0, 0) = 0$,

(ii) $t(a, b) = t(b, a)$,

(iii) $t(a, b) = t(c, d)$ for $a = c$, $b = d$,

(iv) $t(t(a, b), c) = t(a, t(b, c))$.

Definition 3:

A Menger PM-space is a triplet (X, F, t) where (X, F) is PM- space and t-norm t satisfies $F_{p,r}(x+y) \geq t\{F_{p,q}(x), F_{q,r}(y)\}$ for all $x, y \geq 0$ and $p, q, r \in X$.

Note that among a number of possible choices for t, $t(a, b) = \min\{a, b\}$ or simply "$t = \min$" is the strongest possible universal t (cf. [12]).

Schweizer and Sklar [11] have proved that if (X, F, t) is a Menger PM-space with a continuous t-norm, then X is a Hausdorff space in the topology T induced by the family of neighbourhoods

$\{N_p(\varepsilon,\lambda): p \in X, \varepsilon > 0, \lambda > 0\}$ where $N_p(\varepsilon,\lambda) = \{x \in X: F_{x,p}(\varepsilon) > 1-\lambda\}$.

Definition 4:

A sequence $\{u_n\}$ in a probabilistic metric space X is said to converge to u if and only if for each $\lambda \geq 0, x \geq 0$, there exists a positive integer $N(x,\lambda) \in N$ such that

$F_{u_n,u}(x) > 1-\lambda$ for all $n \geq N$.

Or, equivalently $\lim_{n \to \infty} F_{u_n,u}(x) = 1$.

Let F and G denote the distribution functions related to Menger spaces X and Y respectively.

MAIN RESULTS

Theorem 1:

Let (X, F, t) and $(Y, G\ t)$ be complete Menger spaces, where t is a continuous t-norm with $t(x,x) \geq x$ for all x in X. If S is a mapping from Y to X and T is a continuous mapping from X to Y satisfying.

$$F_{STp,STp'}(hx) \geq \min\{F_{p,STp}(x), F_{p',STp'}(x), F_{p,STp'}(\alpha x), G_{Tp,Tp'}(x)\} \tag{1}$$

$$G_{TSq,TSq'}(hx) \geq \min\{G_{q,TSq}(x), G_{q',TSq'}(x), G_{q,TSq'}(\alpha x), F_{Sq,Sq'}(x)\} \tag{2}$$

for all $p, p' \in Y$, where $0 < h < 1$ and $1 < \alpha \leq 2$. Then ST has a unique fixed point z (say) in X and TS has a unique fixed point w (say) in Y. Further, $Tz = w$ ans $Sw = z$.

Proof :

Pick $p_0 \in X$. There exists a point p_1 in X and q_1 in Y such that $(ST)p_0 = p_1$ and $Tp_0 = q_1$. Inductively, we can find sequences $\{p_n\}$ in X and $\{q_n\}$ in Y as follows:

$$(ST)^n p_0 = p_n, T(ST)^{n-1} p_0 = q_n, n = 1, 2, ... \tag{3}$$

From (1),

$$F_{p_n,p_{n+1}}(x) \geq \min\{F_{p_{n-1},p_n}(x/h), F_{p_n,p_{n+1}}(x/h), F_{p_{n-1},p_{n+1}}(\alpha x/h),$$

$$G_{q_{n,} q_{n+1}}(x/h)\}.$$

Taking $\alpha = 1+h$, we have

$$F_{p_n, p_{n+1}}(x) \geq \min\{F_{p_{n-1}, p_n}(x/h), F_{p_n, p_{n+1}}(x), G_{q_{n,} q_{n+1}}(x/h)\}$$

since $F_{p_{n-1}, p_{n+1}}((1+h)x/h) \geq \min\{F_{p_{n-1}, p_n}(x/h), F_{p_n, p_{n+1}}(x)\}$

Therefore, $F_{p_n, p_{n+1}}(x) \geq \min\{F_{p_{n-1}, p_n}(x/h), G_{q_{n,} q_{n+1}}(x/h)\}$

Similarly, from (2),

$$G_{q_n, q_{n+1}}(x) \geq \min\{G_{q_{n-1}, q_n}(x/h), F_{p_{n-1}, p_n}(x/h)\}$$

Therefore by induction,

$F_{p_n, p_{n+1}}(x) \geq \min\{F_{p_0, p_1}(x/h^n), G_{q_{1,} q_2}(x/h^n)\}$ and

$G_{q_n, q_{n+1}}(x) \geq \min\{F_{p_0, p_1}(x/h^n), G_{q_1, q_2}(x/h^{n-1})\}$ for n = 1, 2, ….

Since $h < 1$, $F_{p_n, p_{n+1}}(x) \to 1$ and $G_{q_n, q_{n+1}}(x) \to 1$ as $n \to \infty$. Thus as in [2] (see also Lemma 3 [1]), $\{p_n\}$ and $\{q_n\}$ are Cauchy sequences. Let z and w be their respective limits. Then continuity of T together with (3) implies

$$\lim_n q_n = \lim_n Tp_n = Tz = w \tag{4}$$

Since $\lim p_n = z$ and $\lim q_n = Tz$, there exists an integer $N = N(x, \lambda)$ such that for $x > 0$, $0 < l < 1$,

$$F_{z, p_{n+1}}\left(\frac{1-h}{2h}x\right) > 1 - \lambda, \quad F_{p_n, p_{n+1}}\left(\frac{1-h}{2h}x\right) > 1 - \lambda$$

and $G_{Tz,q_{n+1}}(\frac{1-h}{2h}x) > 1 — \lambda$ for $n \geq N.$ (5)

Now from (1), taking $\alpha = 1 + h$,

$$F_{STz,p_{n+1}}(x) = F_{STz,ST(ST)^n p_0}(x)$$

$$\geq \min\{F_{z,STz}(x/h), F_{p_n,p_{n+1}}(x/h), F_{z,p_{n+1}}(\frac{1+h}{h}x),$$
$$G_{Tz,q_{n+1}}(x/h)\}.$$

Since $F_{z,STz}(x/h) \geq \min\{F_{z,p_{n+1}}(\frac{1-h}{2h}x), F_{p_{n+1},STz}(\frac{1+h}{2h}x)\}$

and in view of $\frac{1+h}{h}x > \frac{1-h}{2h}x$, $\frac{x}{h} > (\frac{1-h}{h})\frac{x}{h}$, the above inequality implies that

$$F_{STz,p_{n+1}}(x) \geq \min\{F_{z,p_{n+1}}(\frac{1-h}{2h}x), F_{STz,p_{n+1}}(\frac{1+h}{2h}x), F_{p_n,p_{n+1}}(\frac{1-h}{2h}x),$$
$$G_{Tz,q_{n+1}}(\frac{1-h}{2h}x)\}$$

Therefore from (5) and in view of $\frac{1+h}{2h}x > x$, we have

$$F_{STz,p_{n+1}}(x) > 1 — \lambda \text{ for } n \geq N$$

implies that $STz = z.$

Hence from (4), we have

$STz = Sw = z.$

To prove the uniqueness of z as a fixed point of ST, let y be another fixed point of ST, then from (1) and (2), for any $x > 0$,

$$F_{z,y}(x) = F_{STz,STy}(x)$$

$$\geq \min\{F_{z,STz}(x/h), F_{w,STy}(x/h), F_{z,STy}(\alpha x/h), G_{Tz,Ty}(x/h)\}$$

$$= \min\{1, 1, F_{z,y}(\alpha x/h), G_{Tz,Ty}(x/h)\}$$

$$= G_{Tz,Ty}(x/h) \text{ since } x < \alpha x/h$$

$$= G_{TSTz,TSTy}(x/h)$$

$$\geq \min\{G_{Tz,TSTz}(x/h^2), G_{Ty,TSTy}(x/h^2), G_{Tz,TSTy}(\alpha x/h^2), F_{STz,STy}(x/h^2)\}$$

$$\geq \min\{G_{Tz,Tz}(x/h^2), G_{Ty,Ty}(x/h^2), G_{Tz,Ty}(x/h^2), F_{z,y}(x/h^2)\}$$

$$= F_{z,y}(x/h^2), \text{ a contradiction.}$$

Therefore z is a unique fixed point of ST.

Similarly, w can be shown to be the unique fixed point of TS.

Theorem 2:

Let $(X, \mathcal{F}, t)$ be complete Menger space, where t is a continuous t-norm with $t(x, x) = x$ for all x in X. If S and T are mappings from X to itself, with T is continuous and satisfying

$$F_{STp,STq}(hx) \geq \min\{F_{p,STp}(x), F_{q,STq}(x), F_{p,STq}(\alpha x), F_{Tp,Tq}(x)\} \tag{6}$$

$$F_{TSp,TSq}(hx) \geq \min\{F_{p,TSp}(x), F_{q,TSq}(x), F_{p,TSq}(\alpha x), F_{Sp,Sq}(x)\} \tag{7}$$

for all $p, q \in X$, $0 < h < 1$ and $1 < \alpha \leq 2$; then ST has a unique fixed point z and TS has a unique fixed point w. Further, $Tz = w$ and $Sw = z$ and if $z = w$ then z is the unique common fixed point of S and T.

Proof :

The existence of z and w follows from Theorem 1. If $z = w$, then z is necessarily a common fixed point of S and T.

Suppose that S and T have another fixed point y. Then from (6) and (7), for any $x > 0$,

$$F_{z,y}(x) = F_{STz,STy}(x)$$

$$\geq \min\{F_{z,STz}(x/h), F_{y,STy}(x/h), F_{z,STy}(\alpha x/h), F_{Tz,Ty}(x/h)\}$$

$$= \min\{F_{z,z}(x/h), F_{y,y}(x/h), F_{z,y}(\alpha x/h), F_{z,y}(x/h)\}$$

$$= F_{z,y}(x/h), \text{ a contradiction.}$$

Hence, S and T have a unique common fixed point

COROLLARY 1. Let (X, f, t) be complete Menger space where t is a continuous t-norm with $t(x, x) > x$ for all x in X. If S is a continuous self map on X such that

$$F_{Sp,Sq}(hx) \geq \min\{F_{p,Sp}(x), F_{q,Sq}(x), F_{p,Sq}(\alpha x)\} \text{ for all } p, q \in X, \tag{8}$$

h $\in (0, 1)$, $\alpha \in (1,2]$; then S has a unique fixed point.

Setting, either $S = I$ (identity map in (7) or $T = I$ in (8), its proof follows from Theorem 1.

The metric version of Theorem 1 is given as follows.

Theorem 3:

Let (X, d) and (Y, ρ) be complete metric spaces. If S is a mapping from Y to X and T is continuous mapping from X to Y safisfying

$$d(STp, STp') \leq h \max\{d(p, STp), d(p', STp'), \frac{1}{\alpha} d(p, STp'), \rho(Tp, Tp')\} \tag{9}$$

$$\rho(TSq, TSq') \leq h \max\{\rho(q, TSq), \rho(q', TSq'), \frac{1}{\alpha}\rho(q, TSq'), d(Sq, Sq')\} \tag{10}$$

for all $p, p' \in X$; $q, q' \in Y$ where $0 < h$ and $1 < \alpha \leq 2$. Then ST has a unique fixed point z (say) in X and TS has a unique fixed point w (say) in Y. Further, $Tz = w$ and $Sw = z$.

The proof of Theorem 3 is based on the fact that, a complete metric space may be regarded as a complete Menger space if we take

$$F_{u,v}(x) = H(x - d(u,v)) \text{ and } G_{u,v}(x) = H(x - \rho(u,v))$$

where

$$H(x) = \begin{cases} 0 & \text{for } x = 0 \\ 1 & \text{otherwise} \end{cases}$$

and $t(a,b) = \max\{a,b\}$. It may be seen that conditions (9) and (10) then imply conditions (1) and (2) respectively.

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 45-50

ELECTRONIC WASTE "E-WASTE" : A NEW ENVIRONMENTAL THREAT

Vinod Kumar* and A.K. Chopra*

(Received 14-09-2007)

ABSTRACT

The use of electrics and electronics has increased many folds since last decade. In developing countries especially in India, life style has changed considerably due to advent of industrial revolution manufacturing electronic gadgets. The problem created by electronic waste (e-waste) has made both developed and developing countries to go for a permanent solution to this mushrooming problem. In India e-waste management, assumes greater significance not only due to the generation of our own waste but also dumping of e-waste from developed countries. As a result e-waste is becoming anthropogenic hazard and generating various human health problems as they are carcinogenic, mutanogensis, causing cancer, neurological, cardiac disorders and genetic deformities also. The disposal of e-waste requires an urgent need of safe disposal and management of this new environmental threat.

Key words : e-waste, anthropogenic hazard, environmental threat.

INTRODUCTION

In the rapid pace of technology, the application of electrics and electronics are increasing day by day. It creates a tremendous revolution in every trend of life such as data processing, communication, entertainment and luxuries. But it generates serious and new anthropogenic environmental hazards because the number of discarded and old equipments and appliances are fastly increasing as its use in new technology. Hazardous waste is waste that causes potential threats to public health or the environment and generally exhibits one or more characteristics. It may be from small company's dry cleaners, automobile repair shops, hospitals, exterminators, photo processing centers and large companies like chemical manufacturers, electroplating companies, and oil refineries. Apart from these products the electronic goods pose severe problems while recycling due to the presence of hazardous substances. E-waste has been increasing rapidly with the rise of the information technology. It is the fastest growing sector of the municipal solid waste. E-waste equals 1% of solid waste on an average in developed countries and is expected to grow to 2% by 2010 [2]. In developing countries, E-waste range from 0.01% to 1%. Industrialized nations like the USA are the largest producer and consumers

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*Department of Zoology & Environmental Science, Gurukula Kangri University Haridwar - 249404 (Uttarakhand)

of electronic goods about 48.5 million computers discarded in the United States each year and 50-80 percent of e-waste collected for recycling is exported from the U.S. to Asia. China had the highest growth in number of computer users per capita in the period 1993-2000. It grew a massive 1052%, compared to a world average of 181%. Asian countries are fastly converting to grave yard of electronic waste because most of the Asian countries are developing; they depend on developed countries like the US for technological support [6]. So these countries like India are turn to disposing sites of e-waste for developed world. India's capital Delhi is emerging as the world's capital for e-waste recycling, a hazardous activity taking place without any regulations, warns a new study. As many as 900 personal computers and 3,500 TV sets are dismantled in the city every day for reuse of their component parts and materials. "Delhi alone gets around 70% of the electronic waste generated in the developed world. In terms of total e-waste produced internally or brought from outside for recycling, Delhi's e-waste weighs between 10,000 and 12,000 metric tones per year and it is estimated that in 2012, it will reach up to 20,000 MT per year" [4, 6]

WHAT IS E-WASTE?

Electronic waste or e-waste is unwanted electrical or electronic appliance that have been discarded by their original users and in most cases it consist of more or less durable products used for data processing, telecommunications or entertainment in households and commercial places-waste are considered dangerous they contain hazardous substances, which pose threat to health of both human and the environment. Thus it is defined as a waste type consisting of any broken or unwanted electrical or electronic appliance. It is a point of concern considering that many components of such equipments are considered toxic and are not biodegradable. [7]

HAZARDOUS CHEMICALS IN E-WASTE

E-waste contains over 1,000 different substances and chemicals, many of which are toxic and are likely to create serious threat to the environment and human health if not handled properly. However, classification of e-waste as hazardous, or otherwise, depends on the amount of hazardous constituents present in it. Some of these are lead, mercury, chromium barium, beryllium and cadmium in different parts of these electrics and electronic devices and equipments such as lead in circuit boards, lead oxide and cadmium in monitor cathode ray tubes (CRTs); mercury in switches and flat screen monitors; cadmium in computer batteries; polychlorinated biphenyls (PCBs) in older capacitors and transformers; and brominated flame retardants on printed circuit boards, plastic casings, cables and polyvinyl chloride (PVC) cable insulation that release highly toxic dioxins and furans when burned to retrieve copper from the wires. Due to the presence of these hazardous chemicals, disposing and recycling of E-waste has serious legal and environmental implications. When computer waste is land filled or incinerated, it causes significant contamination problems. Landfills leach toxins into groundwater and incinerators emit toxic air pollutants including dioxins. So the recycling of computers has serious occupational and environmental implications, particularly when the recycling industry is often marginally profitable at best and often cannot afford to take the necessary precautions to protect the environment and worker health [1,8].

Lead : The severe effects of lead are very well recognized. It was first banned from gasoline in the 1970s. It causes damage to the central and peripheral nervous systems, blood systems, kidney and reproductive system in human. It also affects the endocrine system, and impedes brain development among children. Lead tends to accumulate in the environment and has high acute and chronic effects on plants, animals and micro-organisms. It is used in glass panels and gaskets in computer monitors solder in printed circuit boards and other components.

Cadmium : Cadmium compounds are highly toxic with a possible risk of irreversible effects on human health, and accumulate in the human body, particularly in the kidneys. Cadmium founds in certain components such as SMD chip resistors, infra-red detectors, and semiconductor chips. Cadmium is also a plastics stablizer and some older cathode ray tubes contain cadmium.

Mercury : Mecrucy can cause damage to organs including the brain and kidneys, as well as the foetus. The developing foetus is highly vulnerable to mercury exposure. When inorganic mercury spreads out in the water, it is transformed to methylated mercury which bio-accumulates in living organisms and concentrates through the food chain. It is used in thermostats, sensors, relays, switches (e.g. on printed circuit boards and in measuring equipment) medical equipment, lamps, mobile phones and in batteries. Mercury, used in flat panel displays, will likely increase as their use replaces cathode ray tubes.

Barium : Barium is a soft silvery-white metal that is used in computers in the front panel of a CRT, to protect users from radiation. A short-term exposure to barium has caused brain swelling, muscle weakness, damage to the heart, liver, and spleen.

Beryllium : Beryllium is a steel-grey metal that is extremely lightweight, hard, a good conductor of electricity and heat, and is non-magnetic. These properties make beryllium suitable for many industrial uses, including, electronic applications such as computers. In computers, beryllium is commonly found on mother-boards and "finger clips" as a copper beryllium alloy used to strengthen the tensile strength of connectors and tiny plugs while maintaining electrical conductivity. It is recognized as human carcinogen as exposure to it can cause lung cancer. The primary health concern is inhalation of beryllium dust, fume or mist. Workers who are constantly exposed to beryllium, even in small amounts, and who become sensitized to it can develop what as Chronic Beryllium Disease (beryllicosis), a disease which primarily affects the lungs. Exposure to beryllium also causes a form of skin disease that is characterized by poor sound healing and wart-like bumps.

Hexavalent Chromium (VI): Hexavalent Chromium (VI) can cause damage to DNA and is extremely toxic in the environment. It is used as corrosion protection of untreated and galvanized steel plates and as a decorative or hardener for steel housings. It easily passes through cell membranes and is then absorbed producing various toxic effects in contaminated cells.

Plastics (including PVC): The largest volume of plastics (26%) used in electronics contains a huge amount of PVC. It is used for its fire-retardant properties. Dioxin is released when PVC is burned, which is very toxic to human health. It causes reproductive and development problems, damage to immune system and interface with regulatory hormones.

Brominated flame retradants (BFRs): BFRs are used in the plastic housings of electronic equipment and in circuit boards to prevent flammability. More than 50% of BFR usage in the electronics industry consists of tetra-bromo-bis-phenol (TBBPA), 10% is polybrominated diphenyl ethers (PBDEs) and less than 1% is polybrominated biphenyls (PBB). It disrupts endocrine system functions.

Toners : It is ever-present in computer as peripheral scraps and post consumer. The plastic printer cartridge containing black and color toners. The main ingredient of the black toner is a pigment commonly called, carbon black, commonly known as commercial powder form of carbon. Through inhalation it causes acute effects and may lead to respiratory tract irritation. The International Agency for Research on Cancer has classified carbon black as a class 2B carcinogen, possibly carcinogenic to humans.

Phosphor and additives : Phosphor is an inorganic chemical compound that is used as a coat on the interior of the CRT faceplate. It affects the display resolution and luminance of the images that is seen in the monitor. The hazards of phosphor in CRTs are extremely toxic as its coating contains heavy metals, such as cadmium, and other rare earth metals, e.g. zinc, vanadium, etc. as additives. These metals and their compounds are very toxic.

GENERAL METHODS OF E-WASTE DISPOSAL

Disposing and recycling of e-waste is an uphill task due to the hazards substances which are found in it. Environmentally safe technology is quite expensive. As most recyclers do not have the technology. The e-waste recycling and disposal methods in India, China and Pakistan pollutes the environment as they do open burning of plastic waste and dump the recycled remains in rivers [5]. E-waste is equally disposed in landfills, incinerators or recycled or reused. In landfills, chemicals from e-waste is burnt toxic gases like dioxins (carcinogenic) are released into the air. This can cause diseases like silicosis, respiratory problems and sometimes even death, due to exposure to various toxic metals. The quantity of hazarous waste and the intensity of the toxic nature vary depending on the nature of raw material used and the amount of products produced. Disposing/recycling of this hazardous waste is becoming unavoidable problem to manufacturers and the consumers, as it gets accumulated in larger amount [3]. Some techniques which are used to dispose the e-waste are as:

Landfill : E-waste is piled up and covered with other domestic waste and soil. Landfill method is often done by small business and by domestic users.

Incineration : E-waste is burnt which produces toxic gases like dioxins and Furans. The toxic smoke released into the atmosphere pollutes the air.

Reuse : The electronic equipment goes for slight modification or may be used as such. About 3%-5% of the computers is being reused.

Recycling : E-waste goes for recycling after all the possibility for reuse. It can be used as raw material to other manufacturing industry.

WHY E-WASTE NEEDS MANAGEMENT ?

In the past few years, technology advances in electronics have boosted the economy and improved the general lifestyle of a common man. The ever-growing dependence on electronic products has paved the way for an emerging environment concern called "Electronic Waste". Electronic equipments contain hazardous material, which can affect human health, and environment, if not properly managed." A list of old, outdated computers, laptops, TVs, cellular phones, mp3 players, telecommunications equipment, instruments, toys, and medical devices etc. comes under E-waste. Presently, electrical and electronic equipment contains material that could be recovered and reused for new product development. Electronic and electrical equipment comprise multiple components, some having toxic substances that can affect human health and environment, if not properly managed. Generally, these hazards occur on account of improper recycling and disposal methods. Therefore, the proper handling and management of this new environment threat is urgently need of the time.

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 51-54

ULTRASONIC STUDY OF LUTIDINES IN WATER

Sanjay Kr. Sharma*

(Received 10-10-2007, Revised 07-12-2007)

ABSTRACT

Ultrasonic velocity measurements provide useful information to investigate the intermolecular interactions and the structure of the liquid state. In present paper, it was planned to investigate various lutidines in solutions in water but practically it was found that 3, 4 - and 3, 5 - lutidines remain insoluble in water. Only 2, 4 - lutidine is soluble but at higher concentrations it also shows non - solubility. Therefore the study has been done for 2, 4 - lutidine in water at temperature of 290.16 K. The ultrasonic velocity in the solutions is measured at frequency of 2 MHz, using interferometric technique. The density, viscosity jand refractive index are also measured using a bicapillary Pycnometer, Ostwald viscometer and Abbe's refractometer respectively. Using the measured data various parameters such as van der Waals constant, adiabatic compressibility, Rao number, Specific acoustic impedance and molecular radius are also computed. With the help of these parameters, conclusions regarding the interactions between different sized lutidine and water molecule are drawn.

पुस्तकालय
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गुरुकुल कांगड़ी विश्वविद्यालय

Key Words: Ultrasonics, Binary liquids mixture.

INTRODUCTION

The ultrasonic study of the binary mixtures has drawn large interest [5, 7, 8] due to simplicity of the technique and providing important information regarding molecular behaviour of the liquids. Lutidines, the dimethyl substituted compound of pyridine, have so for been studied in pure [9] and in solutions [2, 4] using different techniques. But with water out of the lutidine series, studies on only 2, 6- lutidine are reported [1]. In this paper the ultrasonic study of the solutions of 2,4-, 3,4-, and 3,5-, lutidine in water at temperature of 290.16 K has been done by measuring ultrasonic velocity (C) at a frequency of 2 MHz using interferometric technique. Along with the velocity, the measurements of density (ρ), refractive index (n_D) and viscosity (η) have also been made. The measured data are used to compute various ultrasonic parameters.

EXPERIMENTAL

The solubility of lutidine in water was experimentally checked by mixing the solutes in water in different proportions and also heating the solutions every time to high temperatures.

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*Department of Physics, D.B.S. College, Kanpur-208006. E-Mail : sksharma777@gmail.com

The 3, 4-and 3, 5- lutidine remain immiscible with water at all the proportion and temperatures while 2, 4 lutidine is soluble only in low concentration. Therefore leavmg 3, 4- and 3, 5- lutidine due to their insolubility only dilute solutions of 2, 4- lutidine in water are studied.

The ultrasonic velocity in the binary mixture of 2, 4- lutidine and water has been measured with an accuracy of ± 0.3 % using the single frequency (2 MHz) variable path interferometer. The density, viscosity and refractive index have been measured by using the pycnometer, Ostwald viscometer and Abbe's refractometer respectively. The measurements were made at 290.16 K. The sample of 2, 4-lutidine (98 % pure) was procured from Fluka (Germany) and water distillation was made in our laboratory. Using the measured data various parameters such as van der Waals constant (b), adiabatic compressibility (β_a), Rao number (R_n), specific acoustic impedance (z), Molar compressibility (B_w) and effective molecular radius (r_m) are evaluated using the known relations [3,6].

RESULTS AND DISCUSSION

The measured data of C, ρ, η and n_D are given in Table 1 and the computed parameters b, a, R_n, z, B_w and r_m are given in Table 2. From an observation of the Table 1 and Table 2 it is seen that the velocity in solutions is larger than that in the pure components. This may be due to the closer packing of the molecules in the solutions. From the computed values of average molecular radii (r_m), it is seen that water molecules are about half in linear size as compared to the lutidine molecules. It is possible that the water molecules occupy some of the free space that would exist in pure lutidine, making the medium denser for passing ultrasonic. The viscosity of the solutions is also larger compared to the viscosity values of pure component. Also the closer packing of molecules in the solutions is confirmed by decreased compressibility. Rao number (R_n) and Molar compressibility (B_w) being the molecular properties, show an increasing trend with mole fraction of lutidine (f_2). The refractive index shows some variations; first it decreases then there is a slight increase at higher concentrations.

The lutidine molecules as well as water molecules have high dipole moments and are likely to form stronger bonds in the solution. It is expected that this strong polar-polar bonding will lead to increased compressibility and ultrasonic wave speed.

ACKNOWLEDGEMENT

The author is thankful to University Grants Commission (U.G.C.) for the financial support in form of Minor Research Project.

TABLE 1: Measured Parameters for 2,4- Lutidine in Water at 290.16 K

f_2 Mole-fraction	C m/sec.	ρ gm/cm^3	η c poise	n_D
0.0000	1472	0.9987	1.082	1.333
0.0500	1554	1.0017	2.606	1.395
0.1009	1603	0.9894	3.972	1.416
0.1923	1610	0.9896	4.901	1.455
1.0000	1434	0.9274	1.237	1.495

TABLE 2: Computed Parameters for 2,4- Lutidine in Water

f_2 Mole-fraction	b cm^3/mol.	$\beta_a \times 10^{11}$ cm^2/dy.	R_n $\times 10^4$g.sec^{-1}cm^{-2}	z	B_w $\times 10^{-8}$ cm	r_m
0.0000	16.33	4.62	952	14.70	540.20	1.174
0.0500	20.53	4.13	1206	15.56	683.76	1.267
0.1009	25.16	3.93	1482	15.86	837.74	1.356
0.1923	33.00	3.89	1932	15.93	1091.49	1.484
1.0000	109.72	5.24	6046	13.30	3402.75	2.215

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Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 55-59

MECHANISM OF WOUND HEALING BY PEMF STIMULATION

P.P. Pathak* and Devendra Singh*

(Received 16-11-2007, Revised 22-12-2007)

ABSTRACT

Reviewing the work on wound healing by pulsed electromagnetic field (PEMF) stimulation, a possible mechanism is proposed. Qualitative explanation for the observed acceleration of wound healing by action of PEMF is also given.

INTRODUCTION

Utilization of electrical and electromagnetic signal for treatment of many diseases has been done for long time[12]. Particularly in wound healing, the direct application of alternating current (ac) and direct current (dc) stimulations is in frequent use. Formally the effect of ac and dc stimulations on wound healing has also been studied by placing electrodes distal and proximal to the wound periphery by stimulating wounds for two hours per day, five days per week up to thirty days[13]. It was found that both types of stimulations reduce healing time and increase perfusion in early phases of healing. It was also found that dc stimulation reduces wound area more rapidly than ac but ac stimulus reduces wound volume slightly more rapidly than dc.

There are also the studies of wound healing by low frequency pulsed electromagnetic fields (PEMF)[4] where the authors found that PEMF with 20 Hz pulse rate was very much effective in promoting wound healing. In all these studies, the physical mechanism by which the external stimulus works has not been discussed except a recent model by Pathak et al. [10].

In present study we want to explore the mechanism of wound healing by PEMF. It is proposed that applied stimulus orients new collagen formation and thus enhances wound healing. The orientation is likely to be different in the cases of dc/ac and PEMF and so the rate of healing is different in these cases.

Human body and bodies of all other living creatures have their own electrical-magnetic energy balance. Since our first appearance on the planet, we have also encountered external electro magnetic fields (emfs). The human body floats in a sea of magneticfields, i.e., those of the earth, moon, sun and other galactic fields[12]. The earth itself has an iron core that generates a halo of electromagnetic energy, reaching far beyond our atmosphere. All life on earth has adapted to and existed in this natural electromagnetic environment for millions of years.

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*Department of Physics Gurukul Kangri University, Hardwar-249404

The body is full of magnetisable materials, such as iron in the haemoglobin of the blood. The biomagnetic fields of the body have been measured using SQUIDS (superconducting quantum interference devices) and found to be 10^5 times weaker than the earth's fields[17]. We are so dependent on the earth's fields that when we leave the planet for prolonged periods, we suffer. Astronauts in space for long periods require artificial magnetic fields to maintain health. In the last one hundred years we have radically altered our environment with human-made emfs with a different waveform, coherence and frequency to natural emfs and their effects on biological systems are only just beginning to be understood. Naturally-occurring emfs do not seem to significantly alter the body's natural electromagnetic balance and are thought to be necessary for its proper functioning[4]. Most concern comes from alternating currents (ac) electricity which create an energy field that is both electrical and magnetic. Electrical fields are of little concern as they do not penetrate the body, while ac magnetic fields penetrate organisms.

Fire and electricity convey heat to a substance but microwaves induce heat within a substance by altering its molecular motion. This altered molecular motion might be the mechanism of abnormal healing. Internal sensations of heat generated by microwaves would not necessarily be detected on the surface. As Oschman[9] so ably recounts in his excellent series of articles on healing energy, Zimmerman[16], observing biofields produced by a healer's hand, recorded a pulsed signal of variable frequency ranging from 0.3 to 30 Hz, with most of the activity in the range of 7 - 8 Hz. The signal emitted is not steady or constant but rather sweeps through a range of frequencies. This range covers the low frequency pulses produced by some pieces of physiotherapy equipment used for soft tissue regeneration etc. Only healers were able to produce the biomagnetic pulses[17].

MECHANISM OF ACTION OF HEALING ENERGY

Healing energy could be carried round the body by:

(i) magnetising the blood, (the iron content of haemoglobin being reported by Pauling[11] as conferring magnetic properties on the blood).

(ii) the ionic current created by the electrolytes in the plasma of the blood (dissolved sodium, chlorides, potassium, bicarbonates).

(iii) proteins which serve as semi-conductors.

(iv) the nerves.

(v) the fascia (connective tissue and sheaths surrounding the spinal cord etc.).

(vi) water.

The human body contains approximately 70% water. Many biological activity of compounds are switched on or off by changes in the concentrations of dissolved ions such as sodium and potassium. Individual water molecules, with their tendency to form hydrogen bonds, also stabilize the structure of proteins, whose action depends crucially on their shape[7].

If the hydrogen bonding angles of one molecule are altered, those in the other molecules might be expected to follow suit. Alteration of bonding angles have been shown to follow healing[3,14]. Sweeping of the body during healing could result in re-magnetising of the blood. This could have the two-fold action of transferring healing energy throughout the body via the circulation to parts where it is needed and of helping to restore particular vibrational frequency of the person receiving healing. Vibration of the body will occur as a whole, at a very minute level, affecting water molecules, individual cells and helping to restore the normal L-field[5,8] and resonant frequency of DNA within the nucleus. It was Linus Pauling in the 1920s who first pointed out that at the heart of the covalent hydrogen bond is resonance[11]. As the hydrogen bond is an integral part of water molecules, enzymes, DNA, all food substances etc., it follows that if healing energy affects this bond it will be able to influence every part of the body, in particular DNA and enzyme activity.

Cells communicate with each other and the vibratory energy will reach within the cell via the cytoskeleton and microtubules. Lakhovsky[6] found that the individual cell has two characteristics capacitance and inductance - which are the elements of a tuned circuit like a radio, the cell must be tuned to the desired frequency, i.e. that necessary to sustain life. As Becker[1] has shown, the requirements for normal differentiation of cells and final shape and size attained by body tissue is dictated by minute (micro) electric currents, which are in turn dictated by the bio-magnetic field of the body. This also relates to healing energy which is thought to produce tiny eddy currents in body tissue.

EXPERIMENTAL STUDIES

Hajizadeh et al.[4] studied the effect of PEMF on wound healing in rats. They used forty eight wistar male rats weighing 200-300 g. These were kept in standard cages and food and water were available to them without any limitation. The room temperature was also kept constant at 20 ± 2°C. Insensibility of the part of the body which was having wound was performed by intraperitoneal injection of examine hydrochloride, 100 mg/kg and pompun, 10 mg/kg, 10 minutes before surgery. After shaving and cleaning, a pair of full thickness incisions (35 mm in length) was made parallel to and at a distance of 1.5 cm on both sides of the dorsal midline of the rat. Then the animals were divided into eight groups viz. the control, sham and six treatment groups (each group having 6 rats). The treatment groups were exposed to PEMF (4×10^{-3} T) of pulse rates of 10,20,40,50,60 and 80 Hz.

The sham exposed animals were exposed inside the restrainer kept inside an unenergized coil (the same condition as the experimental animals). The pulsed electromagnetic field flux direction was parallel to the cuts made on the rat's body. The animals in the control group were wounded like other groups but were kept as such throughout the experiment. The system was fed by a function generator or producing a square wave with 2 ms pulse width. The out put of the function generator was amplified by a home made audio amplifier connected to a coil made of 8400 turns of copper wire with 8 cm (internal diameter) and 3.5 cm in length producing 4×10^{-3} T field effective intensity on the surface of the rat skin.

The field strength was measured by a Hall effect teslameter. The pulsed electromagnetic fields were applied to the treatment groups for half an hour, two times in a day for 10 successive days. The duration of the wound healing was the time taken for full contraction of the wound. After the twenty days of surgery, all the animals (participated experimentally) were killed by an overdose of ether and the tensile strength of each scar was measured.

RESULTS AND DISCUSSION

It seems that PEMF stimulation may orient new collagen formation in a pattern similar to normal skin. Application of electrical stimulation enhances tissue perfusion in early phases of healing more significantly with PEMF stimulation. In this way, the finding of wound healing acceleration by PEMF stimulation above the normal healing without stimulation is confirmed i.e. all the wounds healed rapidly in comparison to non-stimulated wound[5]. The wounds initially increased in both surface area and volume. Following the reach of a maximum value, each wound gradually reduces in size as granulation tissues are formed and fill up the wound. The application of PEMF stimulation affects the reduction of wound area more than reduction of the wound volume. Contraction of the wounds occurres more rapidly in stimulated animal than the non-stimulated animals.

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AN ECOLOGICAL STUDY OF THE PREVALENCE, MEAN INTENSITY AND RELATIVE DENSITY OF THE CESTODE INFECTION IN FRESHWATER FISH, *MASTACEMBELUS ARMATUS* (LACEPEDE)

A.K. Srivastava*, R.K. Khare and V.K. Sahu*****

(Received 29-12-2007)

ABSTRACT

Freshwater fish, *Mastacembelus armatus* (Lacepede) was selected to study the prevalence, mean intensity and relative density of the cestode infection for two successive years (2003-2004 & 2004-2005). The maximum infection occurred in the first half of summer and winter seasons, moderate in second half of summer and winter seasons whereas minimum in rainy season.

Keywords : Prevalence, Mean intensity, Relative density, *Mastacembelus armatus*, River Betwa, Costode.

INTRODUCTION

Mastacembelus armatus is a well known freshwater edible fish. This fish has highly nutritive food value and known to harbour a number of cestode parasites, which causes harmful effect on nutritive and market value. An extensive survey since July 2003 to June 2005 was conducted to study the prevalence, mean intensity and relative density of cestode infection in *Mastacembelus armatus* at river Betwa, district Jhansi (U.P.) India.

MATERIALS AND METHODS

Fresh water fish, *Mastacembelus armatus* were obtained from river Betwa through local fish catchers. Live fishes were anesthetized with the help of chloroform and then dissected quickly. The alimentary canals of the hosts were removed and cut open in normal saline water. Cestode parasites were counted separately in each infection. The prevalence, mean intensity and relative density of costode infection were calculated seasonally and monthly on the basis of following formulae which were given by Morgolis et al. [6].

$$\text{Prevalence} = \frac{\text{Number of hosts infected}}{\text{Numbers of hosts examined}}$$

$$\text{Mean intensity} = \frac{\text{Total number of cestodes obtained}}{\text{Total number of hosts infected}}$$

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*Parasitological Laboratory, Department of Zoology, Bipin Bihari (P.G.) College, Jhansi (U.P.)

$$\text{Relative density} = \frac{\text{Total number of cestodes obtained}}{\text{Total number of hosts examined}}$$

OBSERVATIONS

There are three seasons from ecological point of view *viz.* rainy, winter and summer. The rainy season includes July, August, September and October. Winter season includes November, December, January and February while summer season includes March, April, May and June. Out of 240 hosts examined, only 31 were found infected with 49 cestodes.

Average seasonal variations in the prevalance, mean intensity and relative density of cestode infection in *Mastacembelus armatus* (Lacepede) have been depicted in the Table-1 and fig. 1.

The prevalance of cestode infection was highest (0.212) during summer season and lowest (0.05) in rainy season. The mean intensity of cestode infection was highest (1.9) during winter season and lowest (1.0) during rainy season. The relative density of cestode infection was highest (0.325) during summer season and lowest (0.05) in rainy season.

Average monthwise variations in the prevalence, mean intensity and relative density of cestode infection in *Mastacembelus armatus* (Lacepede) have been depicted in Table- 2 and Fig. 2.

The maximum prevalence (0.35) was recorded in the month of April whereas minimum (O) in August, September, December and January. In rest of the months it ranges in between 0.1 to 0.25. The maximum mean intensity (2.4) was recorded in November whereas minimum (O) in August, September, December and January. In rest of the months it ranges from 1.0 to 1.857. The relative density (0.65) was maximum in the month of April whereas minimum (O) in August, September, December and January. In rest of the months it ranges from 0.1 to 0.6.

DISCUSSION

The food of *Mastacembelus armatus* (Lacepede) comprised of molluscs, crustaceans and their larvae, insects and their larvae etc. and acts as intermediate hosts which is affected by seasonal and monthwise variations. Markov and Rogoza [4] reported that greater helminths infection occurred in spring season. Less [2] reported that the highest incidence of parasitization by helminths occurred in the autumn season in united Kingdom, where insects and other arthropods reappear after winter diapause with the maximum in spring i.e. helminth abundance follows intermediate host abundance. Kinsella [1] reported that parasitic prevalence during summer and rainy seasons and believes that the greater occurrence of arthropods in these seasons is the sole reason for their prevalence. From the available reports thus a strong indication exists that there is a definite correlation between the occurrence of the parasites and their intermediate hosts during the year. Mathur [5] and Lohia [3] reported that the highest prevalence, mean intensity and relative density of cestodes infection during summer season. Pathak [7] reported that the highest prevalence and relative density during summer season while highest mean intensity during rainy season. Singh and Malik [8] reported that the infection percentage increased rapidly from spring to summer season. On the basis of above discussion it can be concluded that the fish, *Mastacembelus armatus (*Lacepede) in river Betwa, district, Jhansi (U.P.) India have lowest immunity during first half of the summer and winter seasons, moderate during second half of summer and winter seasons and highest during rainy season.

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Table- 1 : Average seasonal variations in the prevalence, mean intensity and relative density of cestode infection in *Mastacembelus armatus* (Lacepede)

Season	**No. of hosts**		**Prevalence**	**Number of cestodes obtained**	**Mean intensity**	**Relative density**
	Examined	**Infected**				
Rainy	80	04	0.05	04	1.0	0.05
Winter	80	10	0.125	19	1.9	0.237
Summer	80	17	0.212	26	1.529	0.325

Table -2 : Average monthwise variations in the prevalence, mean intensity and relative density of cestode infection in *Mastacembelus armatus* (Lacepede)

Month/ Years	**No. of hosts**		**Prevalence**	**Number of cestodes obtained**	**Mean intensity**	**Relative density**
	Examined	**Infected**				
July (03-04)	20	2	0.1	2	1.0	0.1
Aug. (03-04)	20	0	0	0	0	0
Sept. (03-04)	20	0	0	0	0	0
Oct. (03-04)	20	2	0.1	2	1.0	0.1
Nov. (03-04)	20	5	0.25	12	2.4	0.6
Dec. (03-04)	20	0	0	0	0	0
Jan. (04-05)	20	0	0	0	0	0
Feb. (04-05)	20	5	0.25	7	1.4	0.35
March (04-05)	20	4	0.2	5	1.25	0.25
April (04-05)	20	7	0.35	13	1.857	0.65
May (04-05)	20	4	0.2	5	1.25	0.25
June (04-05)	20	2	0.1	3	1.5	0.15

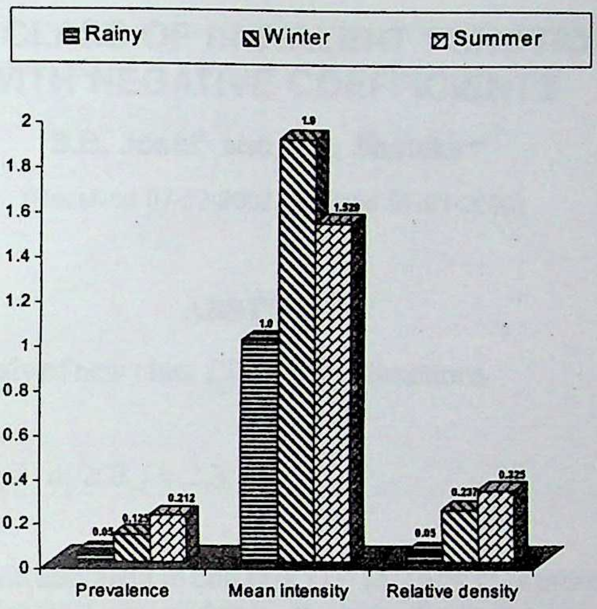

Fig. 1: Average seasonal variations in the prevalence, mean intensity and relative density of helminths infection in *Mastacembelus armatus* (Lacepede)

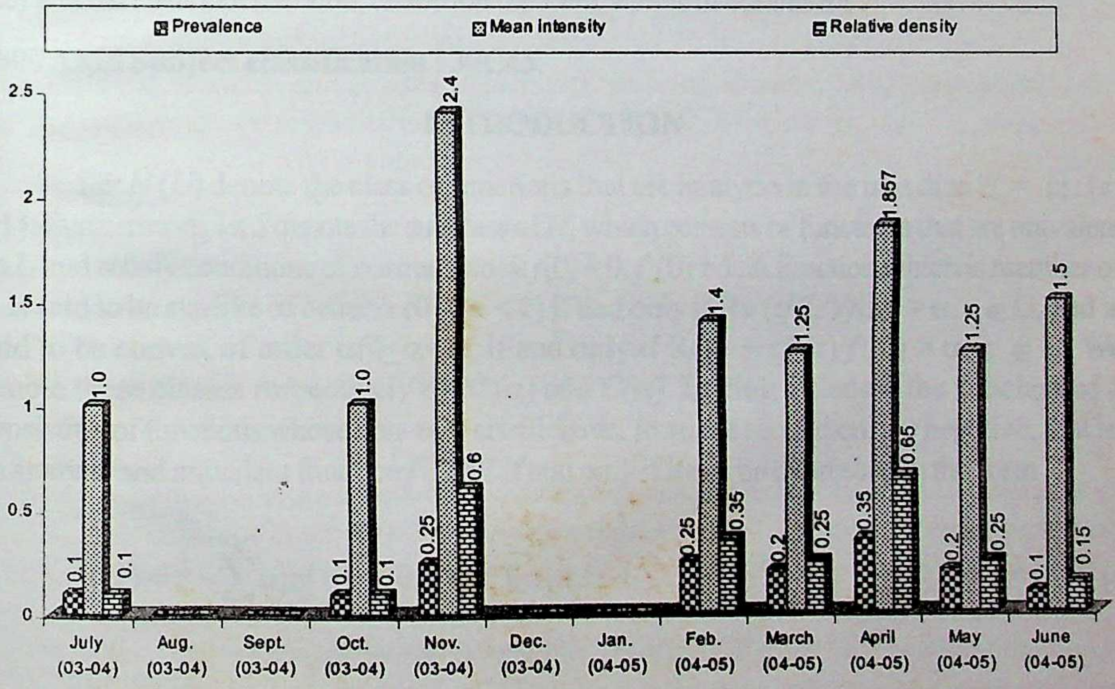

Fig. 2 : Average monthwise variations in the prevalence, mean intensity and relative density of cestode infection in *Mastacembelus armatus* (Lacepede)

Journal of Natural & Physical Sciences, Vol. 21 (1-2) (2007) 67-78

ON A CLASS OF UNIVALENT FUNCTIONS WITH NEGATIVE COEFFICIENTS

S.B. Joshi* and G.D. Shelake**

(Received 07-07-2007, Revised 07-01-2008)

ABSTRACT

A systematic study of new class $T_n(\alpha, \beta, \gamma)$ of functions

$$f(z) = z - \sum_{j=2}^{\infty} a_j z^j \quad a_j \geq 0, j = 2,3\ldots..,$$

which are normalized, and univalent in unit disk $U = \{z : |z| < 1\}$ is presented. Among other result we determine characterization of $T_n(\alpha, \beta, \gamma)$, to obtain sharp results for distortion theorems, radius of convexity and closure theorem.

Keywords. : Starlike function, distortion theorem, radius of convexity.

2000 AMS Subject classification : 30C45.

INTRODUCTION

Let $H(U)$ denote the class of functions that are analytic in the unit disc $U = \{z : |z| < 1\}$. Furthermore, let S denote the subclass of H, which consists of functions that are univalent on U and satisfy conditions of normalization $f(0) = 0, f'(0) = 1$. A function which is member of S, is said to be starlike of order α $(0 \leq \alpha < 1)$ if and only if $\mathrm{Re}\,(zf'(z)/f(z)) > \alpha$, $z \in U$, and is said to be convex of order $\alpha (0 \leq \alpha < 1)$, if and only if $\mathrm{Re}(1 + zf''(z)/f'(z)) > \alpha$, $z \in U$. We denote these classes respectively by $S^*(\alpha)$ and $C(\alpha)$. Letting T denote the subclass of S consisting of functions whose non-zero coefficients, from the second on, are negative; that is, an analytic and univalent function f is in T if and only if it can be expressed in the form

$$f(z) = z - \sum_{j=2}^{\infty} a_j z^j \quad a_j \geq 0, j = 2,3,\ldots\ldots \tag{1.1}$$

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*Department of Mathematics, Walchand College of Engineering, Sangli, (M.S.) India 416415.
Email : joshib@hotmail.com.
**Department of Mathematics, Willingdon College Sangli, (M.S.) India 416 416
Email : gdshelake@rediffmail.com

We denote N_0, the set of all non-negative integers ($N_0 = (0,1,2,....)$).

Definition 1[2]:

We define the operator $D^n : S(U) \rightarrow S(U)$, $n \in N_0$, by

$$D^0 f(z) = f(z) = z + \sum_{j=2}^{\infty} a_j z^j .$$

$$D^1 f(z) = zf'(z) = z + \sum_{j=2}^{\infty} j a_j z^j .$$

$$D^n f(z) = D(D^{n-1} f(z)) = z + \sum_{j=2}^{\infty} j^n a_j z^j .$$

Definition 2:

Let $\alpha \in [0,1)$, $\beta \in (0,1]$, $\gamma \in (1/2,1]$, and let $n \in N_0$, we define, the class $S_n(\alpha, \beta, \gamma)$ of n-starlike function of order α, type β and γ by.

$$S_n(\alpha,\beta,\gamma) = \{ f \in H(U) : f(0) = f'(0) - 1 = 0 \;\; and \left| J_n(f,\alpha,\gamma;z) \right| < \beta, \, z \in U \}$$

where

$$J_n(f,\alpha,\gamma;z) = \frac{\left(\dfrac{D^{n+1} f(z)}{D^n f(z)} - 1 \right)}{2\gamma \left(\dfrac{D^{n+1} f(z)}{D^n f(z)} - \alpha \right) - \left(\dfrac{D^{n+1} f(z)}{D^n f(z)} - 1 \right)}, \;\; z \in U.$$

We note that $S_0(\alpha,1,1)$ is class of starlike functions of order α and $S_1(\alpha,1,1)$ is class of convex functions of order α and $S_n(\alpha,1,1)$ is class of n-starlike function of order α defined in [2]; S_n $(\alpha, \beta, 1)$ is class of n-starlike function of order α of type β defined [3]. We also note that the class $S_0(\alpha,\beta,\gamma)$ is introduced and studied by Kulkarni [1]. We note that not much attention was given to such type of work for last so many years. Motivated by aforementioned work of various authors, we now present here

$$T_n(\alpha,\beta,\gamma) = S_n(\alpha,\beta,\gamma) \cap T$$

and study basic properties, such as characterization, distortion theorems, radius of convexity and closure theorem.

COEFFICIENT INEQUALITIES

Theorem 1:

Letting $\alpha \in [0,1)$, $\beta \in (0, 1]$, $\gamma \in (1/2,1]$, and $n \in N_0$, The function f of the form (1.1) is in $T_n(\alpha,\beta,\gamma)$ if and only if

$$\sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma\ j-2\gamma\alpha)]a_j \le 2\beta\gamma(1-\alpha). \tag{2.1}$$

The result (2.1) is sharp.

Proof:

We suppose that (2.1) holds. Then we have

$$|J_n(f,\alpha,\gamma;z)| = \left| \frac{\left(\dfrac{D^{n+1}f(z)}{D^n f(z)}-1\right)}{2\gamma\left(\dfrac{D^{n+1}f(z)}{D^n f(z)}-\alpha\right)-\left(\dfrac{D^{n+1}f(z)}{D^n f(z)}-1\right)} \right|$$

$$= \left| \frac{\sum_{j=2}^{\infty} j^n a_j(1-j)z^j}{2\gamma(1-\alpha)z-\sum_{j=2}^{\infty} j^n a_j(1-j+2\gamma j-2\gamma\alpha)z^j} \right|.$$

Letting $|z| = 1$, we have

$$\left|\sum_{j=2}^{\infty} j^n(j-1)a_j z^j\right| - \beta\left|2\gamma(1-\alpha)z-\sum_{j=2}^{\infty} j^n(1-j+2\gamma j-2\gamma\alpha)a_j z^j\right|$$

$$\le \sum_{j=2}^{\infty} j^n[(j-1)+\beta(1-j+2\gamma j-2\gamma\alpha)]a_j - 2\beta\gamma(1-\alpha) \le 0$$

where we used (2.1).

From the last inequality we deduce

$$|J_n(f,\alpha,\gamma;z)| \le \beta, \quad |z| = 1.$$

Hence $|J_n(f,\alpha,\gamma;z)| < \beta, \quad z \in U$ and $f \in T_n(\alpha,\beta,\gamma)$.

Conversely we assume that $f \in T_n(\alpha,\beta,\gamma)$. Then

$$|J_n(f, \alpha, \gamma; z)| < \beta \qquad z \in U. \tag{2.2}$$

For $z \in [0, 1)$ (z real) the inequality (2.2) can be written

$$-\beta < \frac{\sum_{j=2}^{\infty} j^n (j-1) a_j z^{j-1}}{2\gamma(1-\alpha) - \sum_{j=2}^{\infty} j^n (1-j+2\gamma j-2\gamma\alpha) a_j z^{j-1}} < \beta. \tag{2.3}$$

We note that $E(z) = 2\gamma(1-\alpha) - \sum_{j=2}^{\infty} j^n (1-j+2\gamma j-2\gamma\alpha) a_j z^{j-1} > 0, z \in [0,1)$, because $E(z) \neq 0$ for $z \in [0, 1)$ and $E(0) = 2\gamma(1-\alpha) > 0$. Upon clearing the denominator in (2.3) and letting $z \to 1$ through real values, we deduce

$$\sum_{j=2}^{\infty} j^n (j-1) a_j \le 2\beta\gamma(1-\alpha) - \beta \sum_{j=2}^{\infty} j^n (1-j+2\gamma j-2\gamma\alpha) a_j .$$

Thus

$$\sum_{j=2}^{\infty} j^n [j-1+\beta(1-j+2\gamma j-2\gamma\alpha)] a_j \le 2\beta\gamma(1-\alpha).$$

The external function are

$$f_j(z) = z - \frac{2\gamma\beta(1-\alpha)}{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]} z^j \qquad j = 2,3,\ldots.. \tag{2.4}$$

Corollary 1.

If $f \in T_n(\alpha, \beta, \gamma)$ then

$$a_j \leq \frac{2\gamma\beta(1-\alpha)}{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]} \quad j=2,3,\ldots\ldots$$

The result is sharp and the extremal functions are given by Eqn. (2.4).

DISTORTION THEOREM

Theorem 2:

Let $\alpha \in [0,1)$, $\beta \in (0,1]$, $\gamma \in (1/2,1]$, and let $n \in N_0$, if $f \in T_n(\alpha, \beta, \gamma)$, then for $0 < |z| = r < 1$, we have

$$r-\frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}r^2 \leq |f(z)| \leq r+\frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}r^2 \quad (3.1)$$

with equality for

$$f(z)=z-\frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}z^2 \quad (z=\pm r).$$

Proof:

Form (2.1) we have

$$2^n[1+\beta(4\gamma-2\gamma\alpha-1)]\sum_{j=}^{\infty} a_j \leq \sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)] \leq 2\gamma\beta(1-\alpha)$$

$$\sum_{j=2}^{\infty} a_j \leq \frac{\gamma(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}. \quad (3.2)$$

Thus for $0 < |z| = r < 1$

$$|f(z)| \leq r+\sum_{j=2}^{\infty} a_j r^j \leq r+\frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}r^2.$$

And

$$|f(z)| \geq r - \sum_{j=2}^{\infty} a_j r^j \geq r - \frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} r^2 .$$

Thus (3.1) follows.

Theorem 3:

The disk $|z| < 1$ is mapped onto a domain that contains the disk

$$|w| < 1 - \frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]}$$

by any $f \in T_n(\alpha, \beta, \gamma)$.

Proof:

The result follows upon by letting $r \to$ in theorem (3.1)

Theorem 4:

Let $\alpha \in [0,1)$, $\beta \in (0,1]$, $\gamma \in (1/2,1]$. and let $n \in N_0$, if $f \in T_n(\alpha, \beta, \gamma)$, then for $0 < |z| = r < 1$, we have

$$1 - \frac{2\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} r \leq |f'(z)| \leq 1 + \frac{2\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} r . \tag{3.3}$$

Equality holds for

$$f(z) = z - \frac{\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} z^2 \quad (z = \pm r).$$

Proof:

From (2.1) we have

$$2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)] \sum_{j=2}^{\infty} j a_j \leq 2\gamma\beta(1-\alpha)$$

$$\sum_{j=2}^{\infty} j a_j \leq \frac{2\beta\gamma(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} .$$

Thus

$$|f'(z)| \le 1 + \sum_{j=2}^{\infty} ja_j r^{j-1} \le 1 + \frac{2\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} r.$$

And

$$|f'(z)| \ge 1 - \sum_{j=2}^{\infty} ja_j r^{j-1} \ge 1 - \frac{2\gamma\beta(1-\alpha)}{2^{n-1}[1+\beta(4\gamma-2\gamma\alpha-1)]} r.$$

Thus (3.3) follows.

RADIUS CONVEXITY FOR FUNCTION IN T_n (α, β, γ).

Theorem 5:

If the function $f(z) = z - \sum_{j=2}^{\infty} a_j z^j \quad a_j \ge 0, j = 2,3,\ldots$ is in T_n (α, β, γ), then f is convex in the disk

$$|z| < r = r(\alpha,\beta,\gamma,n) = \inf_j \left(\frac{j^{n-2}[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}{2\gamma\beta(1-\alpha)} \right)^{\frac{1}{j-1}} \quad (j = 2,3\ldots)$$

This result is sharp, with the extermal functions as given in (2.4).

Proof:

It suffices to show that $\left|\frac{zf''(z)}{f'(z)}\right| \le 1$ for $|z| \le r(\alpha,\beta,\gamma,n)$. We have

$$\left|\frac{zf''(z)}{f'(z)}\right| = \left| \frac{\sum_{j=2}^{\infty} j(j-1)a_j z^{j-1}}{1-\sum_{j=2}^{\infty} ja_j z^{j-1}} \right| \le \frac{\sum_{j=2}^{\infty} j(j-1)a_j |z|^{j-1}}{1-\sum_{j=2}^{\infty} ja_j |z|^{j-1}}.$$

Thus $\left|\frac{zf''(z)}{f'(z)}\right| \leq 1$ if

$$\sum_{j=2}^{\infty} j(j-1)a_j|z|^{j-1} \leq 1 - \sum_{j=2}^{\infty} ja_j|z|^{j-1}$$

or

$$\sum_{j=2}^{\infty} j^2 a_j |z|^{j-1} \leq 1. \tag{4.1}$$

According to Theorem 1,

$$\sum_{j=2}^{\infty} \frac{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}{2\gamma\beta(1-\alpha)} a_j \leq 1$$

Hence (4.1) will be true if

$$j^2|z|^{j-1} \leq \frac{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}{2\gamma\beta(1-\alpha)} \tag{4.2}$$

Solving (4.1) for |z|, we obtain

$$|z| \leq \left(\frac{j^{n-2}[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}{2\gamma\beta(1-\alpha)}\right)^{\frac{1}{j-1}} \quad (j = 2,3\ldots). \tag{4.3}$$

Setting $|z| = r(\alpha,\beta,\gamma,n)$ in (4.3), the result follows.

CLOSURE THEOREMS

Let the function $f_k(z)$ be defined for $k \in \{1,2,3,\ldots,m\}$, by

$$f_k(z) = z - \sum_{j=2}^{\infty} a_{j,k} z^j, (a_{j,k} \geq 0) \tag{5.1}$$

for $z \in U$.

Now we shall prove the following result for the closure of function in the class $T_n(\alpha, \beta, \gamma)$.

Theorem 6:

Let the function $f_k(z)$ defined by (5.1) be in class $T_n(\alpha, \beta, \gamma)$ for every $k \in \{1,2,3,...,m\}$. Then the function $F(z)$ defined by,

$$F(z) = z - \sum_{j=2}^{\infty} b_j z^j, \ (b_j \geq 0) \tag{5.2}$$

is a member of class $T_n(\alpha, \beta, \gamma)$, where

$$b_j = \frac{1}{m}\sum_{k=1}^{m} a_{j,k} \quad (j=2,3,....). \tag{5.3}$$

Proof:

Since $f_k(z) \in T_n(\alpha, \beta, \gamma)$, it follows from Theorem 1, that

$$\sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]a_{j,k} \leq 2\beta\gamma(1-\alpha)$$

for every $k \in \{1,2,3,...., m\}$. Hence,

$$\begin{aligned}\sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]b_j &\leq \sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]\left(\frac{1}{m}\sum_{k=1}^{m} a_{j,k}\right)\\ &= \frac{1}{m}\sum_{k=1}^{m}\left(\sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]a_{j,k}\right)\\ &\leq \frac{1}{m}\sum_{k=1}^{m} 2\gamma\beta(1-\alpha) = 2\gamma\beta(1-\alpha).\end{aligned}$$

which (in view of Theorem1) implies that $F(z) \in T_n(\alpha, \beta, \gamma)$.

Theorem 7:

The class $T_n(\alpha, \beta, \gamma)$ is closed under convex linear combination.

Proof:

Let the function $f_k(z)$ defined by (5.1) be in class $T_n(\alpha, \beta, \gamma)$ for every $k \in \{1,2,3,...,m\}$, it is suffices to prove that the function

$$H(z) = \lambda f_1(z) + (1-\lambda) f_2(z), (0 \le \lambda \le 1) \tag{5.4}$$

is also in class $T_n(\alpha,\beta,\gamma)$. Since, for $0 \le \lambda \le 1$,

$$H(z) = z - \sum_{j=2}^{\infty} \{\lambda a_{j,1} + (1-\lambda) a_{j,2}\} z^j, \qquad (0 \le \lambda \le 1). \tag{5.5}$$

We observe that

$$\begin{aligned}
&\sum_{j=2}^{\infty} j^n [j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]\{\lambda a_{j,1} + (1-\lambda) a_{j,2}\} \\
&\qquad = \lambda \sum_{j=2}^{\infty} j^n [j-1+\beta(1-j+2\gamma j-2\gamma\alpha)] a_{j,1} \\
&\qquad + (1-\lambda) \sum_{j=2}^{\infty} j^n [j-1+\beta(1-j+2\gamma j-2\gamma\alpha)] a_{j,2} \\
&\qquad \le \lambda 2\gamma\beta(1-\alpha) + (1-\lambda) 2\gamma\beta(1-\alpha) = 2\gamma\beta(1-\alpha).
\end{aligned}$$

with the aid of Theorem 1. Hence $H(z) \in T_n(\alpha, \beta, \gamma)$. This completes the proof of Theorem 7.

Theorem 8:

Letting

$$f_1(z) = z,$$

and

$$\tag{5.6}$$

$$f_j(z) = z - \frac{2\gamma\beta(1-\alpha)}{j^n [j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]} z^j. \tag{5.7}$$

we have $f(z) \in T_n(\alpha, \beta, \gamma)$ if and only if it can be expressed in the form

$$f(z)=\lambda_1 f_1(z)+\sum_{j=2}^{\infty}\lambda_j f_j(z) \tag{5.8}$$

where $\lambda_j \geq 0 (j=1,2,3,\ldots.)$ and $\lambda_1+\sum_{j=2}^{\infty}\lambda_j=1.$ (5.9)

Proof:

Suppose that

$$f(z)=\lambda_1 f_1(z)+\sum_{j=2}^{\infty}\lambda_j f_j(z)$$

$$=z-\sum_{j=2}^{\infty}\lambda_j \frac{2\gamma\beta(1-\alpha)}{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}z^j. \tag{5.10}$$

Since

$$\sum_{j=2}^{\infty} j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]\frac{2\gamma\beta(1-\alpha)}{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}\lambda_j$$

$$=2\beta\gamma(1-\alpha)\sum_{j=2}^{\infty}\lambda_j$$

$$\leq 2\beta\gamma(1-\alpha)$$

By Theorem 1; $f \in T_n(\alpha, \beta, \gamma)$

Conversely, we suppose that $f \in T_n(\alpha, \beta, \gamma)$.

Since

$$a_j \leq \frac{2\gamma\beta(1-\alpha)}{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]} \quad j=2,3\ldots.$$

Setting

$$\lambda_j=\frac{j^n[j-1+\beta(1-j+2\gamma j-2\gamma\alpha)]}{2\gamma\beta(1-\alpha)}a_j \tag{5.11}$$

and

$$\lambda_1 = 1 - \sum_{j=2}^{\infty} \lambda_j \ .$$

we have

$$f(z) = \lambda_1 f_1(z) + \sum_{j=2}^{\infty} \lambda_j f_j(z) \ .$$

This completes the proof of Theorem 8.

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